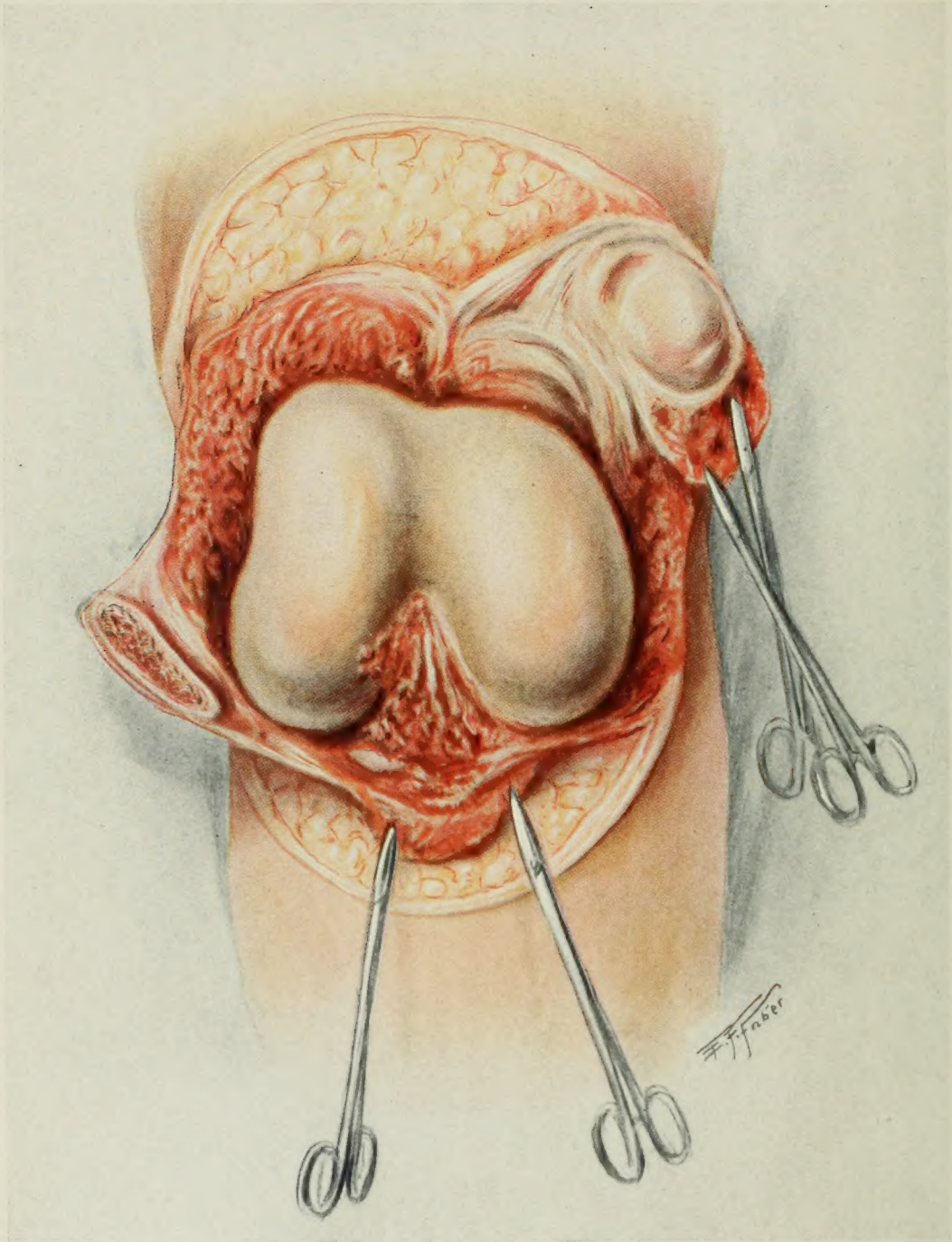


Frederic Graham



Hypertrophic villous synovitis of knee. Note the congestion and velvety appearance of the synovial membrane. Note also the elongated villi between the femur and tibia, where they are pinched during motion of the joint.

See page 62

INTERNATIONAL CLINICS

A QUARTERLY

OF

ILLUSTRATED CLINICAL LECTURES AND
ESPECIALLY PREPARED ORIGINAL ARTICLES

ON

TREATMENT, MEDICINE, SURGERY, NEUROLOGY, PÆDIAT-
RICS, OBSTETRICS, GYNÆCOLOGY, ORTHOPÆDICS,
PATHOLOGY, DERMATOLOGY, OPHTHALMOLOGY,
OTOLOGY, RHINOLOGY, LARYNGOLOGY,
HYGIENE, AND OTHER TOPICS OF INTEREST
TO STUDENTS AND PRACTITIONERS

BY LEADING MEMBERS OF THE MEDICAL PROFESSION
THROUGHOUT THE WORLD

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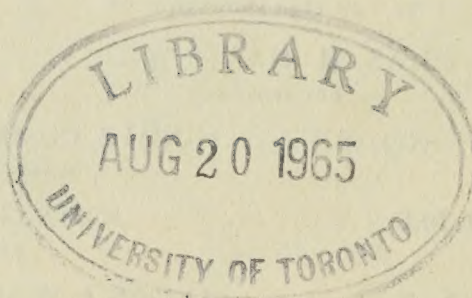
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Clinics

CLINICAL EVIDENCE OF ADRENAL INJURY IN INFECTIONS

CLINIC OF DR. A. S. BLUMGARTEN,
Lenox Hill Hospital, New York.

It is being more and more appreciated that the endocrine glands play an important role in immunity. Numerous observers notably A. Marie,¹ A. Garibaldi,² J. Koopman,³ and others have shown that the thyroid and especially the adrenal glands play an important rôle in the mechanism of immune processes. Although the source of the production of various immune bodies has not been definitely established, yet there is a good deal of evidence accumulating which seemingly indicates that the various endocrine glands perform an important function in this mechanism. Recognition of the presence of various antibodies gives us evidence of biologic substances resulting from the immunological reaction. At the same time, however, this process is manifested by definite clinical evidence, which if viewed in the proper light, can be demonstrated to be the clinical manifestation of the immune reaction.

The reaction of the endocrine glands in infections can occur in two ways: first, as the clinical evidence of an immunological response to the infection, and second, as evidence of injury to one of several of the endocrine glands, either during the infection itself or after the infection has subsided.

To get a proper perspective of the whole problem let us for a

¹ MARIE, A.: Du mode d'action de l'Adrenaline sur les toxines bacteriennes, *Annales de l'Institut Pasteur*, Paris, 1919, 33, pp. 645-656.

² GARIBALDI, A.: Thyroïde et Immunité acquise. Sur l'influence de la thyroïdectomie (chez le lapin) sur le formation de sensibilisatrices hétérohemolytiques d'immunisation. *Comptes rendus. Société de Biologie*, Paris, 1920, 83, pp. 15-16.

³ KOOPMAN, J.: The Influence of the Thyroid Gland on the Formation of Antibodies, *Endocrinology*, vol. ii, No. 3, July-September, 1919.

moment consider some of the clinical features of common infections from this point of view. It is known to any good clinician that individuals differ in the intensity of their reaction to an infection. Very frequently the more intense the reaction as indicated by the intensity of the clinical signs the better is the prognosis. For example, a patient suffering from pneumonia who has a moderately high temperature, flushed face, rapid bounding pulse, bright expression of the eyes, etc., frequently does better than the patient whose response is not so marked. Comparing this reaction to the symptoms of a definite endocrine syndrome, for instance, it appears that this general evidence of acute infection corresponds quite closely, with the exception of the temperature, to hyperthyroidism. Consequently we may reasonably assume that the thyroid gland plays an important rôle in this reaction. On the other hand it is well known that tympanites, profound asthenia, general exhaustion, and low blood-pressure are of sinister significance in an infection, be it pneumonia, typhoid or any of the acute exanthemata. This reaction is always explained as evidence of a vasomotor paralysis of the splanchnic terminals of the sympathetic. Yet many of us appreciate to-day that this explanation is still *sub judice*.

It is fairly well established that the adrenal glands are the sensitizers of the sympathetic nervous system. Now it is definitely established that the clinical syndrome of tympanites, asthenia, general exhaustion, and low blood-pressure are all evidences of sympathetic paralysis. This sympathetic paralysis, however, may result not only from an actual injury to the sympathetic nerve terminals themselves but also from a lack of sympathetic sensitization resulting from the disturbance in the function of the adrenal glands. Furthermore, we know that infections such as scarlet fever frequently cause serious injury to the kidney, and since the infectious agent of scarlet fever, whatever this may be, affects the kidney by circulating in the blood stream, why should the scarlet fever poison leave uninjured such a highly important and adjacent organ as the adrenals that are certainly supplied by the same blood as the kidneys. It might be argued, of course, that the toxins, or whatever the infectious agent of scarlet fever is, have a specific affinity for the kidneys. Yet it is just as logical to assume that the scarlet fever toxin has no specific affinity for the kidney cells, but that we are better able to recognize

the injury to the kidneys clinically, than we are the injury to the adrenals. I venture to state also that the injury to the adrenals results in more serious consequences to the patient than injury to the kidneys. The adrenal injury probably results in death much more frequently, a death which we usually explain as being due to a profound toxæmia. This death results so quickly that evidence of adrenal injury is overlooked. Furthermore, the explanation of the profound toxic death in an acute infection as being due to cardiac failure does not appear to be so evident, as cardiac failure is usually due to toxic degeneration of the heart muscle. But such toxic degeneration has rarely been proved as an isolated phenomenon other than as part of generalized muscular degeneration resulting from the toxæmia. We know also that patients having much more extensive degeneration of the heart muscle than occurs in the hearts of the acute death from an infection do not have such profound disturbances in cardiac function. Consequently it seems more likely that the acute death in an infection is rather the result of overcoming the immune processes of the body by the infectious agent or its toxins than by degeneration of the heart muscle.

If the clinical syndrome of sympathetic paralysis noted above were the result of the specific affinity of the infectious agent for the sympathetic terminals or the sympathetic nervous system, we should get at the same time evidences of injury to other parts of the nervous system, because if the infectious agent has a specific affinity for these terminals it must be a chemical one and depend upon the chemical structure or composition of the nervous tissue, as all nerve tissue is more or less of the same chemical structure no matter where or what its distribution. Since these cases showing the sympathetic paralysis syndrome are relatively free from evidence of injury to other parts of the nervous system it seems rather unlikely that the lesion in this condition is in the sympathetic nerves. A good deal of evidence, however, shows that the adrenal glands play an important rôle not only in the economy of the individual but also in immunity. Since evidence of sympathetic paralysis results when the infection is getting the upper hand it is much more rational to explain the sympathetic deficiency syndrome on the basis of a real injury of the adrenal glands themselves by the infectious agent or their toxins. Clinically, however, it has been difficult to recognize evidence of adrenal injury

although injury of the kidneys by the same generic poison can be recognized readily because of the more definite and accurate methods.

The recognition of adrenal injury has been difficult because it is frequently masked by other symptoms. The endocrine organs, act as a system. Functionally they are correlated; some are synergistic, others antagonistic to one another. Injury to one endocrine gland frequently results in a disturbance of the endocrine balance, and this disturbs the normal relationship. As a result we have symptoms referable to other glands or compensatory symptoms; for example, cases of functional deficiency of the adrenal glands following infections frequently do not manifest themselves by the classical clinical syndrome but by symptoms of hyperthyroidism or by evidence of vagotonia. This is due to the fact that hyperthyroidism is not a uniglandular syndrome but is evidence of hyper function of the thyroid and the adrenal medulla. Now there is a good deal of evidence which goes to show that in a great many ways the adrenal cortex and the adrenal medulla are functionally antagonistic to one another. G. A. Friedman has especially called attention to this.

In order to appreciate this interrelationship let us consider for a moment the clinical and well-known syndrome of real organic disease of the adrenals namely: Addison's disease. It is well known that Addison's disease is characterized essentially by profound asthenia, gastric symptoms, low blood-pressure, extensive pigmentation, slow movements and slow mental activity. There is a good deal of evidence to show that the asthenia and the pigmentation are due to the involvement of the adrenal cortex, while the low blood-pressure is very likely due to the adrenal medulla. The mental sluggishness which is very characteristic of Addison's disease is probably due to failure in the reciprocal action between the cerebrum and the adrenal cortex. We have embryological evidence that such a relationship exists in the fact that an anencephalic monster has an absence of the adrenal cortex. It is also well known and this point has been especially emphasized by Osler that one of the most important and characteristic symptoms of hyperthyroidism and of Grave's disease is profound asthenia. Indeed this is what most of these patients complain of first. Pigmentation is also a very common condition in Grave's disease. It is especially evident in Grave's disease during pregnancy. Consequently we may regard these symptoms of asthenia and pigmentation

occurring in Grave's disease as possible evidence of deficient activity of the adrenal cortex. Conversely when we have evidence of adrenal cortical injury we have a disturbance in the apparent reciprocal action between the adrenal cortex, medulla, and cerebrum, and thus we get hyperthyroidism resulting from a primary injury to the adrenals. A post-infectious neurasthenia, therefore, is really a hyperthyroidism resulting from adrenal injury during infection. It seems very likely that in such cases there is a real injury to the adrenals particularly to the adrenal cortex as a result of the initial infection. It is, of course, interesting from the endocrine and general immunologic standpoints as to why one individual should manifest this particular syndrome and another one should not.

It is, of course, well recognized that individuals differ and that this difference may be racial, anatomic, psychologic, or physiologic. And if we add the evidence of the Schick test and other immunologic reactions there is also immunologic differences. Now we know fairly definitely that the endocrine glands play an important rôle in growth and development and very likely also in the psychological and physiological manifestations of the individual. Consequently whatever individual characteristics the patient has they are the result of his own singular endocrine physiology. If we study individuals therefore, and classify them in accordance with the well-known classical endocrine syndromes we may group individuals into types according to the endocrine gland which apparently dominated the growth and development of the individual and which dominates as well the physiology and psychology. Hence we may group individuals into definite types: The so-called tropisms of Fraenkel, Kaplau and others—the thyrotrope, adrenotrope, gonadotrope. It should be definitely understood that this classification of individuals according to their endocrine domination does not mean that they are diseased in most instances or in need of treatment. It simply indicates that their growth, development, physiology and psychology have been patterned along characteristic lines. It is simply their own singular normal. It is important to appreciate this because the endocrine system may be considered the system which balances the function of the body, and that there are numerous possible variations from the so-called normal which are still within normal limits but that these variations

are important in determining the individual's singular reactions not only to infections but to many other factors in life.

It should not be understood that because the individual manifests evidence of a singular endocrine function that the symptoms he suffers in a given disease are necessarily due to a disturbance in the endocrine glands. The individual may be a thyrotrope and suffer from a disease that has no relation to his tropism. An adrenotrope may suffer from conditions that have no bearing whatsoever on his dominating adrenal activities. On the other hand however, in many instances there seems to be and it frequently can be demonstrated that there is a relationship between the symptoms and his tropism. Here I want to emphasize that the occurrence of endocrine reactions in infections for instance are not necessarily due to a specific affinity between the endocrine organ and the infectious agent, but are the result of the individual's unique endocrine domination. In any machine or mechanism it is usually the most highly sensitive parts that get out of order easily, consequently if the individual shows evidence of domination of certain organs in his physiology, these organs are more sensitive and functionate more frequently. It seems rational then that these organs would show evidence of injury from extraneous causes or infections before other organs; especially under conditions in which the dominating organ comes into play. Thus we see that the specific affinity between an extraneous substance or infectious agent for an endocrine organ is not necessary. The fact that an endocrine organ plays the dominating rôle in the endocrine physiology exposes it to frequent injury from infection or other extraneous elements.

The adrenal glands may respond to an infection in another manner. Since it is fairly well established that the thyroid and adrenal apparatus plays an important rôle in immunity, infections while stimulating the production of various specific and nonspecific immune reactions may also show clinical evidence of their production. Infections may manifest themselves by various phenomena indicating either primary or compensatory disturbance in the function of the ductless glands. But this clinical syndrome, however, is really an expression on the part of the patient of his reaction to the invading organism. Let us forget for a moment this explanation of the reaction of the endocrine glands to infection and merely review some of the known clinical signs of infections. Every good clinician

knows that certain systemic infections particularly chronic infections may manifest themselves by symptoms of irritation of the nervous system for instance, or by symptoms which may coincide closely with a frank endocrine syndrome such as hyperthyroidism. It is well known that tuberculosis may begin with symptoms referable to the various abdominal viscera such as the stomach; producing gastric symptoms or that disturbance in metabolism such as constant loss in weight, or with tachycardia, with profuse sweating or headaches or nervousness and very frequently with emotional outbursts. On the other hand the literature records numerous cases of hyperthyroidism resulting from focal infections of the tonsils, teeth, or elsewhere in the body. Indeed McCarrison's patient and thorough work on the problem of endemic goitre has led him to the conclusion that it is a result of a chronic infection probably from the large intestine.

I wish to point out that these numerous symptoms can be put together into a definite endocrine syndrome which is the expression of some infection. I do not wish to convey the idea, however, that every time such a syndrome occurs that it is due to an infection. But we must bear the syndrome in mind as a possible explanation of an infection. It is up to the physician, however, to try to find the focus, and if such a one is found we can then interpret the symptoms correctly. Frequently the causative infection can only be demonstrated subsequently, because the endocrine reactions may be the earliest expressions of it. But we must consider all the clinical data in determining the condition and their proper interpretation is dependent upon good clinical judgment. We cannot, therefore, divorce endocrinology from general medicine. It does not constitute a specialty but is merely one phase of general medicine; an important phase of the patient's constitutional reaction. Endocrine reactions in medicine play the same rôle as fever or the pulse, in other words it is merely a study of the patient's reaction to injuries, to discord, to sympathetic or unsympathetic environment, etc.

This afternoon I wish to present a number of cases to illustrate the rôle of the adrenal glands in various infections. I realize that at the present time we have to rely largely upon clinical data for the recognition of the adrenal disturbance. Yet it is noteworthy that the progress in the study of the ductless glands has come almost

entirely from the clinical studies of Addison, Graves, Pierre Marie and others.

If we take into account the fact that there is a well-established interrelationship between the various glands and that we have a number of more or less exact methods for determining functional disturbances of the endocrine glands such as basal metabolism, the Goetch test, etc., you see that it is quite possible not only to establish the absence or presence of a definite syndrome, but also to recognize a disturbance of the normal interrelationship. Let me illustrate the application of these tests in determining disturbances of endocrine function. The Goetch test for example, is considered a fairly accurate test for the determination of hyperthyroidism if interpreted properly. The increased basal metabolism is generally accepted as a most accurate guage of hyperthyroidism and conversely a diminished basal metabolism indicates definite hypothyroid activity. Consequently if we have symptoms of emotional outbursts, nervousness, tremors, tachycardia, loss of weight as a syndrome, and this syndrome is corroborated by an increase in the basal metabolism level or by a positive Goetch test we may consider that the patient is suffering from hyperthyroidism. Now how shall we determine whether this is a reaction to an infection or compensatory to injury of another gland. The other clinical data in the clinical course of the case can help us in this regard. If we can demonstrate a systemic or focal infection at the time it is reasonable to assume this to be the cause. On the other hand the symptoms may be merely compensatory phenomena to a lesion or disturbance in function in the adrenal cortex. We can then corroborate the latter assumption by the presence of other well-known symptoms of adrenal functional disturbance such as Sergeant's line, asthenia, low blood-pressure, history of an acute infectious disease having a predeliction for the adrenals, etc.

ADRENAL INJURY FOLLOWING SCARLET FEVER

CASE I.—B. F., a young married man of 30, was seen in consultation with Dr. Healy in February, 1921, with the following history: In November he had an acute attack of scarlet fever which lasted all the month of December, at the end of which time he recovered without complications or evidence of injury to the kidney except for an occasional slight albuminuria. A few weeks before I saw him

he began to complain of intense weakness in the legs so that he was unable to walk or undergo any exertion. He became fatigued very easily, suffered from sleeplessness and became very nervous. He developed epigastric pain coming on about a half hour after eating. He had been constipated.

There was nothing in his past history or in his family history at all relevant to his condition.

The patient was a rather thin, nervous man, very apprehensive and anxious as to his condition. He had a profuse distribution of hair over his chest, abdomen, forearms, and lower extremities. He had also large, brown pigmented areas over the lumbar region, chest and abdomen, but no rash. There was a very marked Sergeant's line. There was no general glandular enlargement. The scalp was covered with abundant black curly hair. There was no exophthalmos, but the corneae were bright, the pupils wide, and reacting to light and accommodation. The teeth were in good condition and there was no increase in the interdental spaces. The palate was high and arched; the tonsils were not enlarged; the thyroid gland was not enlarged. His lungs showed no evidence of any abnormality. His heart was not enlarged. His pulse rate was 96. There was no arterial thickening in any of the palpable or visible vessels. His blood-pressure was 95 systolic and 65 diastolic. The abdomen was not distended; there were no areas of tenderness or rigidity to be felt. The liver, spleen and other abdominal viscera were absolutely normal. The stomach was markedly dilated, the lower portion extending to midway between the umbilicus and pubis. There was a marked splash. There was tenderness on pressure in the epigastric region and also along the lumbar regions. Examination of the nervous system showed no evidence of any organic lesion. The knee jerks were slightly exaggerated; there was slight tremor of both hands. The basal metabolism was plus 36. The urine examination was negative. The blood count showed a slight secondary anemia.

This patient gave evidence of tachycardia, slight tremor, and nervousness following an infection—scarlet fever. There was no exophthalmos or enlargement of the thyroid gland. The slight tremor and increased basal metabolism and tachycardia we may consider as evidence of a mild hyperthyroidism. He also has evidence of gastric

symptoms, but the low blood-pressure and profound asthenia and Sergent's white line are also evidence of adrenal injury. Consequently we may consider the evident hyperthyroidism as compensatory to adrenal injury resulting from his recent attack of scarlet fever because the symptoms occurred immediately after the attack of scarlet fever.

As to the probable mechanism for the occurrence of the symptoms. We know that the adrenals are the great sympathetic sensitizers, we know that the abdominal viscera are supplied by branches from the autonomic and sympathetic nerves, and since there is insufficient sensitization of the sympathetic nerves on account of the adrenal deficiency we get an over-activity of the vagotonic branches with the production of the gastric symptoms. The nervous symptoms are probably the result of functional dissociation between the adrenal and cerebral cortex, and the hyperthyroidism is probably the result of the disturbed relationship between the adrenals and the thyroid.

Recently Marine and Barman have been able to produce hyperthyroidism experimentally in animals by freezing the adrenal cortex.

Now it is interesting to note that this patient has a profuse hair distribution over his body pigmentation, large canines, etc., all characteristics which are believed to indicate the dominant adrenal activity, and since the adrenal glands are probably highly sensitized in this individual they are the glands which suffered from his acute infection resulting in the clinical syndrome for which he came under observation.

ADRENAL INJURY IN THE COURSE OF AN ACUTE EXACERBATION OF A CHRONIC POLYARTHRITIS

CASE II.—V. Z., a young bookkeeper, 19 years of age, came under observation at the Lenox Hill Hospital in April, 1920, complaining chiefly of pain and swelling in the joints, particularly of the hands. For the past two and a half years the patient has had arthritis which began after a tonsillectomy which was done for repeated attacks of sore throat followed by acute attacks of pain and swelling in the joints. The first attack lasted three months with which the patient was in Bellevue Hospital and apparently cured. Since that time the patient has had frequent repeated attacks for which he has been treated in various hospitals. The present attack began about two months before admission, starting with pain, swelling and tenderness in the hands and then involving the shoulders and finally

the joints of the lower extremities. There are no cardiac symptoms. The patient had measles in childhood but no other illness of any sort other than an attack of erysipelas three months before the onset of this attack. He denies venereal disease. His parents are living and well. There is no history of rheumatism in the family although one brother had recurrent attacks of tonsillitis; otherwise his history is negative.

The patient is a young man who is very uncomfortable, complaining of pain in his joints and considerable limitation of motion and deformity largely due to pain. The skin is pale. He has no rash. His scalp is covered by abundant rather thin dark brown hair. He has no exophthalmos. The pupils are round, equal react to light and accommodation. The teeth are in good condition. There is no evidence of infection. The tongue is coated. The pharynx is congested. Both tonsils are absent. The thyroid is not enlarged. The chest is normal in shape and contour. The lungs are normal. The heart is slightly enlarged. The sounds are regular and of good force and muscular quality. There is a low systolic murmur, heard best at the apex and transmitted to the left axilla. The pulse is regular and of good force and muscular quality, 92. The blood-pressure is systolic 110 and diastolic 70. There is no evidence of arterial thickening. The abdomen is not distended. There is no tenderness and no rigidity. The liver is not enlarged. The lower border of the spleen is felt two fingers' breadth below the costal margin. Otherwise the abdominal findings are negative. The extremities show both shoulders, wrists, fingers and ankles swollen. The fingers are spindle-shaped and there is an inflammatory process in most of the joints which are extremely tender but not red. Indeed there is a suggestion of bony proliferation around the joints of the fingers which was not verified by X-ray examination, except for an occasional slight lipping in the radius and ulna. The patient was confined to bed with this subacute inflammation of the joints. The urine examination was negative except for an occasional trace of albumin and an occasionally hyaline cast. The blood count was: W. B. C., 7400; polymorphonuclears, 72 per cent.; lymphocytes, 27; basophiles, 1, and hemoglobin, 70 per cent. The Wassermann was negative. The patient was in the hospital from May 6, 1920, to July 6, 1920. He ran a temperature ranging from 102 to 102 1-2 F., more or less intermittent. About

the beginning of June the temperature began to show a tendency to increase and the joints became more swollen and extremely painful. On June 6th he complained of tremendous weakness which had been increasing from day to day for about a week before that. He developed tremendous abdominal distension and showed a markedly positive Sergent's line. The blood-pressure was then systolic 76 and diastolic 38.

On the basis of profound asthenia, extremely low blood-pressure, tympanites and positive Sergent's line we considered that the patient was suffering with acute adrenal insufficiency as a result of the same infection causing the acute polyarthrititis. The profound asthenia and low blood-pressure were characteristic and the tympanites we considered to be further evidence of sympathetic paralysis as a result of the adrenal injury. This condition was resistant to treatment with pituitrin and adrenal injections. The temperature gradually subsided, the swellings in the joints gradually improved and the patient left the hospital without any elevation in temperature but with a residual stiffness and swelling in the joints, but free from acute symptoms.

ADRENAL INJURY IN THE COURSE OF TYPHOID FEVER.

CASE III.—P. E., a young lady clerk of 22 years, admitted to the Lenox Hill Hospital in July, 1920, complaining of general weakness and soreness in the abdomen. About two weeks before admission the patient began to have malaise. About three days later she had a chill followed by fever and sweating. She had pain over the chest and felt as though she had a cold but never coughed. She developed soreness over the abdomen and chest which had gradually grown worse. Her appetite was poor; she was constipated; her mouth felt dry. She grew progressively weaker.

Except for whooping cough in childhood her past and family histories have been entirely negative. The patient was well-nourished, lying quietly in bed, and quiet apathetic. There was no rash on the skin, and no pigmentation or unusual hair distribution. The scalp was covered by an abundant growth of hair. The pupils of the eyes were round and reacted to light and accommodation. The nose and ears were normal. The breath had a mouse-like odor. The teeth were in good condition. The thyroid was not enlarged. There was no

general glandular enlargement. The chest was normal in size and shape. The lungs were normal. The heart was not enlarged. There was a slow rhythm and good muscular quality. There were no murmurs. The pulse was regular, slow rhythmic, and there was no evidence of arterial thickening. The abdomen was slightly distended and moderately tender to pressure, especially on the right side. The spleen was not felt. There were no rose spots. The patient ran a continuous temperature on admission, with slow pulse. There was abdominal distention and tenderness and general apathy and malaise. The most probable diagnosis was typhoid fever. The spleen, however, was never enlarged and there were never any rose spots. The blood culture, also, failed to reveal any typhoid bacilli. The stool and urine examination did not show any typhoid bacilli. Although we never were able to verify the diagnosis of typhoid fever, on account of the continuous elevation in temperature with the absence of any definite findings to indicate any other cause and the profound asthenia, we considered the case clinically as one of typhoid fever.

About two weeks after admission the patient gradually developed abdominal distention and a marked Sergent's line. The blood-pressure dropped to systolic 90 and diastolic 64. We therefore considered this evidence of adrenal injury in the course of typhoid fever.

The patient went through a very stormy illness with severe hemorrhages necessitating several blood transfusions. There was evidence of marked tenderness and rigidity in the right lumbar region extending over to the abdomen and there was a question whether she had a perforation, but this condition gradually improved without surgical intervention, though it is probable that this was a perforation which had localized itself and drained into the intestinal tract. But the point I wish to emphasize in this case is the fact that the occurrence of the tympanites associated with asthenia and the drop in blood-pressure is a clinical syndrome for which there is a good deal of evidence to indicate that it is the result of adrenal involvement.

CASE.—H. W., a young physician of 29, came under observation in November, 1919, with the following history: For the last week he had a general inaptitude for work and felt very tired. He had severe frontal headache. For the past two days he had a sore throat. There was nothing at all relevant in his past or family history. The onset of his illness began with a chill.

Examination showed a temperature of 104.5°F.; pulse of 90. There was no rash on the skin. There was an extensive distribution of hair over the chest, abdomen, back and extremities. There was no Sergent's line. There was no general glandular enlargement. The nose and ears were normal. The pupils of the eyes were round, equal and reacted to light and accommodation. The teeth were in good condition, the canines being rather long. The throat was markedly congested, especially along the anterior pillars, resembling the characteristic congestion of an influenza. The thyroid was not enlarged. The chest was normal in shape and contour. The lungs and heart were normal. The abdomen was not distended and there was no tenderness and no rigidity. The liver and spleen were not enlarged. The extremities were normal.

The history, apart from the prodromal malaise and lassitude, is of course suggestive of the sudden onset of an acute infection, possibly gripe. Indeed we considered the case as one of gripe or influenza, but under constant observation the temperature persisted and the pulse was rather slow without developing the usual concomitant signs that go with influenza, so we had to abandon the idea of influenza and begin to think of typhoid fever, particularly as the temperature persisted and the pulse was rather slow. We could, however, find no corroborative evidence except a slight leucopenia. Examination of the urine and stools was negative and the Widal test was entirely negative. The spleen could not be felt. About ten days after the patient came under observation a few rose spots appeared on the abdomen and a few kept on appearing during the subsequent days. A blood culture was taken, but no typhoid bacilli could be found. The blood culture was repeated on four or five occasions but no typhoid bacilli were ever found. About the second or third week of the disease the patient became more or less delirious, the pulse was more rapid and the clinical typhoid appearance became more characteristic. The persistence of the temperature, however, for so long a time with absence of other evidence to account for it naturally led us to consider the case as typhoid fever, but throughout the course of the disease we never could corroborate this diagnosis either by blood culture, by stool or urine culture. On one occasion, however, we obtained a slightly suggestive Widal test.

The spleen was never enlarged and the only other clinical corroborative evidence was the few rather suggestive rose spots.

However, for the purpose of this discussion it is not so relevant to establish the definite diagnosis of typhoid fever because the condition we are about to describe can occur in any infection. Suffice it to say that we were dealing with a patient who had an acute infection with a continuous temperature with an occasional suggestive sign of typhoid fever, and so we will consider the patient as suffering from typhoid fever.

About the third week of the illness the asthenia increased and the abdomen became more markedly distended. On examination, in addition to the signs of distention and associated abdominal tenderness a definite Sergent's line could be made out. The blood-pressure, which formerly was 120-75 was now 90-50. The blood examination showed no change in the leucocyte count or differential count. There was no tenderness or rigidity or blood in the stools so we felt pretty certain we were not dealing with a perforation or with a hemorrhage. In the ordinary clinical sense one would say that the patient had tympanites as a complication of typhoid fever. But this merely states that the patient has a certain symptom but gives no clue as to the underlying pathology that is responsible for this except that it is due to a more or less general and profound toxemia. But in view of the fact that this tympanites occurred with a gradually developing and increasing asthenia and with the development of a Sergent's line which was previously not present together with a change of the blood-pressure from 120-75 to 90-70, and in view of the fact that these signs are believed to be more or less characteristic of adrenal insufficiency, we felt certain that the underlying pathology of this complication was probably a real adrenal injury produced by the typhoid infection, or whatever the infection was. He was given injections of pituitrin with adrenalin. In two or three days the tympanites subsided and the asthenia improved, and the blood-pressure increased as well.

This condition which we have just described in the course of typhoid fever is perhaps one of the commonest conditions that is met with. I am not presenting it as something new or startling in typhoid fever, but rather to indicate a definite and possibly more logical explanation of the condition. Since it is well known that asthenia, low blood-pressure and the Sergent's line are indicative of adrenal in-

sufficiency whether they occur spontaneously or in the course of an infection, if they occur in the course of an infection the infectious agent may be responsible for them. Now I believe that tympanites is an equally important symptom of adrenal deficiency. This was formerly explained as being due to vasomotor paralysis but it seems to me much more likely to be due to adrenal deficiency thereby producing defective sympathetic sensitization resulting in distention of the intestines. However, since we know the lesions of typhoid fever are in the intestines the occurrence of tympanites is not so readily associated with the adrenal injury. I believe the other associated symptoms of the condition occur more frequently than is supposed, particularly as no efforts are made in most instances to establish the presence or absence of other signs.

ADRENAL INJURY IN THE COURSE OF PULMONARY TUBERCULOSIS

CASE IV.—M. V., a rather elderly man, 59 years of age, came under observation in October, 1920, with the following history: He was perfectly well until last April when he had a bad cold. This lasted about a week and was accompanied by severe diarrhoea. Both of these conditions improved in a few weeks. The cough persisted, however, and was accompanied by expectoration and a progressive loss of weight. He went to Atlantic City for a week and then to the mountains for a time without improvement. He was then told that he had tuberculosis. What he complained of chiefly, however, and for which he came under observation was cough, expectoration, and progressive weakness and loss of weight. Lately he had been perspiring very profusely. In 1900 he had had typhoid fever and colitis in 1903. About sixteen years ago he was operated upon for cancer of the tongue. The tongue was removed as well as the cervical glands, and he has never had any recurrence since. For the last nine years he has had diabetes. He never smokes but he takes snuff, and except for three or four cups of coffee daily his habits are average.

His father died at the age of 77 years as the result of an operation for the removal of the prostate. His mother died at the age of 68 years of heart failure. One brother died in 1918 of pernicious anemia.

Physical examination shows a moderately emaciated, neurotic

old man. His skin is dry, there is no Sergent's line and no glandular enlargement. There is considerable hair distribution over the chest, arms, both shoulders and slightly on the abdomen. The nose is normal. The ears show chronic thickened retracted drums. The eyes show marked arcus senilis. The teeth are absent. The throat is congested and there is considerable purulent secretion in the retropharyngeal fossa. The larynx is normal. There is a small stump of the tongue remaining.

The chest is pigeon-breasted. The lungs show dullness over the left apex and supraclavicular region. Posteriorly and extending forward to about a finger's breadth above the level of the clavicle was an area where the breathing was amphoric and accompanied by occasional ronchi. At the right base there was an occasional friction fremitus. The abdomen was not distended; there were no areas of tenderness or rigidity. The stomach was dilated, the lower border extending to the level of the navel. The blood-pressure was systolic 150, diastolic 85. The heart was moderately rapid and the pulse about 90. There was no elevation of temperature.

The urine examination showed 3.2 per cent. sugar; sp. gr. 1025; otherwise it was negative. The sputum showed a moderate number of tubercle bacilli. The blood count showed a moderate degree of secondary anemia.

This patient, of course, shows definite clinical evidence of pulmonary tuberculosis with an active lesion in the left apex, probably with cavity formation. He was advised to go away but he absolutely objected to any change from his routine life and persisted in attending to his business while taking the usual precautions against tuberculosis. He was overfed and spent most of his time in the open air. About the early part of December he began to complain of profound weakness and abdominal distention. This persisted and on December 16 his blood-pressure had changed from 150 systolic and 85 diastolic to 120 systolic and 70 diastolic. At the same time, on stroking the skin a well-defined Sergent's white line was manifest. His asthenia gradually became worse so that he was unable to attend to business and he was confined for the most part either in a chair or part of the day in bed. During this time there was no change in the signs of the lung. There was no change in the pulse rate and no elevation of temperature, so that except for the asthenia we could not really state

that his toxemia was increasing. The abdominal distention gradually grew worse in spite of all efforts at treatment. The Sergeant's line was still present. The blood-pressure was gradually diminished during this time from 120 to 110, 100, and 90 systolic. A urine examination showed a reduction in the sugar content. There was no evidence of acidosis present at any time as indicated by the CO_2 combining power.

This patient, suffering with diabetes and pulmonary tuberculosis developed during the course of his infection the clinical syndrome of asthenia, abdominal distention and low blood-pressure, and a Sergeant's white line, all of which we may regard as evidence of injury to his adrenals. This syndrome in the course of his infection we believe was due to an acute adrenal insufficiency probably the result of his tuberculosis.

SUMMARY

I have attempted to present a number of cases to show adrenal injury or adrenal insufficiency occurring in various infections. This condition may occur in any infection. The cases presented show adrenal insufficiency in scarlet fever, acute rheumatic fever, typhoid fever and tuberculosis. The adrenal insufficiency may occur either in the course of the infection as in some of the cases I have described or following the infection as in the case of scarlet fever above noted. The clinical syndrome which we may regard as indicating adrenal insufficiency is profound asthenia, low blood-pressure, and abdominal distention indicating sympathetic paralysis, and the Sergeant's white line. In post-infectious adrenal insufficiency we may have evidence of disturbance of the endocrine interrelationship. Thus we may get a compensatory hyperthyroidism or autonomic gastric symptoms or so-called post-infectious neurasthenia.

The recognition that insufficiency of the adrenal glands is the basis for this syndrome in infections at once suggests very definite therapeutic conclusions, but practically there is quite a hiatus between the determination of the lesion and its treatment. Of course the natural deduction would be that if we have adrenal insufficiency all we have to do is to supply the patient with suprarenal extract and the syndrome disappears. But while this can occasionally be done, in many instances such treatment is disappointing. This, however, does not disprove the etiology of the condition because therapeutic results should

not be taken as proving physiological or pathological facts. In some instances giving suprarenal extract gives very good results, but the difficulty in the use of suprarenal extract is that frequently the preparations used are inert because adrenal substances oxidize very readily and the methods of its manufacture vary so much. In the majority of instances the processes of manufacture are carried on under conditions which permit of very rapid oxidation. On the other hand we can frequently obtain good results by treating the patient with other methods on the basis of adrenal injury as we understand it. For instance, general tonic treatment, and overfeeding frequently result in improvement of the symptoms. Evidence of adrenal insufficiency suggests iron, the use of iron and strychnine and the feeding of fresh animal foods which contain a great deal of the active adrenal elements. I am referring, of course, to adrenal insufficiency occurring in the course of chronic infections or following an infection. During the course of an acute infection treatment should be carried out to increase the sympathetic tone. This can be done by the injection of pituitrin and adrenalin. In post-infectious adrenal insufficiency characterized by neurasthenic symptoms with compensatory thyroid overactivity or vagotonic gastric symptoms good results may be obtained occasionally by giving small doses of suprarenal extract, provided the preparation is reliable, together with the feeding of fresh animal products and treating the gastric symptoms by diet and alkalies. The compensatory hyperthyroidism may be treated like any hyperthyroidism, with complete mental and physical rest, relaxation, overfeeding with carbohydrates, etc. The vagotonic gastric symptoms may be treated either by supplying adrenal extract to increase the sympathetic tone or by diminishing the vagotonia with large doses of atropin or benzyl benzoate.

I want to again emphasize the fact that in spite of the evidence pointing to adrenal injury adrenal extract does not always work in these cases due to the fact that the present methods of manufacture of adrenal extract do not produce substances that can replace the physiologic adrenal hormones as they occur and are produced in the body. When we understand more about the production of these substances and can produce them in such form that they can replace the physiologic hormone, the ideal treatment would be to supply the proper adrenal hormone.

SURGERY OF THE LARGE BOWEL

A CLINIC HELD BEFORE A GROUP OF INVITED SURGEONS, AT THE PORTLAND SURGICAL HOSPITAL
PORTLAND, OREGON. FEBRUARY 24, 1921.

By ROBERT C. COFFEY, M.D.

AFTER we thoroughly study the principles underlying the treatment of a given condition, the technic is a mere mechanical procedure. There is as much difference between a surgeon and a surgical operator as there is between an architect and a carpenter. The analogy is complete. A surgeon who simply does an operation because he has heard of some famous man doing this particular type of operation, or because he has read it in a standard text-book, without looking into the reason for the steps of the operation, based upon a knowledge of the diseased condition for which the operation is done, is simply a carpenter surgeon. Given a definite job to do, he may do it more skillfully than the surgeon. Just as a skilled carpenter may hang a door or make a joint better than the architect.

This does not mean, however, that a thinking man would rather have a carpenter surgeon operate on his family. It is a question of what to do and the reason for doing it that tells in results, chiefly. We should, therefore, discuss principles more than technic.

We have available this morning a group of cases which will serve as illustrations of a number of fundamental principles underlying the surgical treatment of lesions of the large intestine.

There are three forms of disease to be considered:

1. Simple ulcerative colitis.
2. Tuberculous disease of the colon.
3. Cancer.

Several of the most fundamental principles apply in common to all these conditions.

ULCERATIVE COLITIS

The first patient I wish to show you is one who has been symptomatically cured of ulcerative colitis. In this connection I wish to pay tribute to the late Dr. John Young Brown, of St. Louis. He

taught us more than anyone else to use ileostomy for the purpose of giving a complete rest to a diseased condition of the large bowel. He taught us that a patient may live in perfect comfort and health with such a fistula. That such a fistula is far more comfortable than a fistula of the colon and gives complete rest to the colon where therapy or rest chiefly is to be applied. He taught us that there is relatively very little odor connected with such a fistula.

Some years ago I visited his clinic and he showed me one woman who had an ileostomy opening which he had made nine years before, for tuberculosis of the cæcum. The patient was in perfect condition, had been able to make her living by heavy work, such as washing, and was not greatly disturbed by the fistula, insisting that she had never felt the need of its closure sufficiently to give up the time and money necessary to have it closed.

CASE I.—This first patient I present came to me four years ago. She was, at that time, forty years of age; had been ill for fourteen years with chronic dysentery. She had been treated by the best medical men in our section of the country; had suffered intolerable pain; was a complete invalid; unable to do her work and care for her home; was very thin, and presented altogether, a picture of despair. I told her of the results we had had with opening the bowel and leaving it open for a year or more. This was not particularly encouraging to her, but she had suffered so long and so much that she eagerly grasped the opportunity to change her condition.

A fistula was made by the technic which is described in the pictures, about six inches above the ileo-cæcal valve. At the time of this operation large ulcerative areas were discovered in the cæcum and transverse colon. Apparently it was simple ulcerative colitis and did not impress us with the idea that it was tuberculosis. The patient immediately began to improve and in the course of a year had become perfectly healthy, weighed more than she had ever weighed in her life and fully able to do all her housework and care for her family. The fistula was somewhat annoying, so she decided to come down and see if I would close it. I advised her to wait for some time longer. From time to time she would write, asking if I did not think it would be possible to close the fistula. I kept putting her off all the time, with the idea of giving the ulcerated area a chance of complete cure. In the meantime the intestine was being washed out

once a week, followed by an injection of sweet oil, which had been recommended by Dr. John Young Brown, for some reason. I have not used the sweet oil in my later cases. After a little more than two years she returned. I opened the abdomen, cut off the proximal ileum just above the fistula and connected it with the pelvic colon, still leaving the ileostomy opening for therapeutic use in case of necessity. She went home in perfect health and never had a recurrence of her trouble. Finally, about a year later she came back to have the ileostomy closed. It may be that we were excessively cautious in this case, but the patient is here, as you will see, in perfect health. There is probably not a healthier, stronger woman in Portland.

I had another young lady, who agreed to come this morning, but I found that I had more cases than I could use so I told her I would simply tell you about her.

CASE II.—Fourteen years ago she was brought to me by Doctor Baar, of Portland, with a definite ulcerative colitis and very severe anemia. She was about the age of puberty. With the best medical treatment that could be offered she was gradually going down. She was, therefore, turned over to me by Doctor Baar for surgical treatment.

I had not learned of Doctor Brown's method of treating these cases at the time, therefore, brought the appendix up through the rectus muscle, inserted a tube and used the appendicostomy wound for irrigation and other forms of therapy. She began to improve. She soon arrived at the stage of womanhood and was fairly healthy. After two years we cauterized and destroyed the mucous membrane and thereby closed the appendicostomy opening. Six years later she developed a very violent dysentery, intolerable pain and rapid emaciation. By this time she was a patient of Doctor Sears, who referred her to me again for surgical treatment.

We opened the abdomen and found practically all of the ascending and transverse colon and even the descending colon in a very acutely inflamed, ulcerative condition. There was no part of the large intestine in a healthy state except the rectum. We brought the ileum out through the right rectus muscle. The patient made rapid recovery from her acute condition and within two months was back on her feet again, wearing a Delatour bag, shown in the pictures.

An interesting side-light on this case was that being very prominent socially, she might have been seriously embarrassed by this. But as a matter of fact, she went regularly to all the balls and was not in any way deterred on account of this fistula. There was no odor that anyone was able to detect. She wore this bag for two years. We finally opened the abdomen, cut off the ileum, planted it lower down in the pelvic colon and left the ileostomy opening for about three years. Finally, she being perfectly well and there having been no call for use of the fistula for therapeutic purposes, we closed the fistula. She is now perfectly well and has not had a symptom for several years.

We have other cases of this kind which we could present, but these, I think, serve the purpose to illustrate our point. These cases were, in our opinion, not tuberculosis. Complete rest was the cure.

The same principle of rest, however, seems to be just as successful in tuberculous cases.

TUBERCULOSIS OF THE INTESTINE

CASE III.—Fifteen years ago a young woman of sixteen came to me with an incurable fecal fistula of the cæcum. The patient had been operated upon for appendicitis. The appendix had been removed and proved to be tuberculous. The end of the cæcum, which the surgeon thought, at the time, was simply an inflamed cæcum, proved to be a tuberculous cæcum. He had on two or three different occasions attempted to close the fistula but failed. I attempted to close it, after I had devised and described the extra-peritoneal method of closing a fecal fistula, but in each instance found that the disease seemed to advance to the surface. In other words, the fistula was also tuberculous. The abdominal wall became tuberculous.

I finally opened the abdomen, cut off the ileum and planted its proximal end low down in the pelvic colon. The patient had mild diarrhœa for sometime but began to improve and soon regained her normal health. Curiously enough she was one of those rare cases where ascending peristalsis does not take place. Later pictures following bismuth meals showed that there was no back-flow into the cæcum. Notwithstanding this, there was a certain amount of discharge and the fistula was exceedingly slow in healing. We never attempted to close it again but it closed about nine or ten years after this. She is in perfect health. This was my first experience

with rest for tuberculosis of the bowel. Since that time I have had a number of most gratifying results.

Last week a beautiful, healthy young woman came into my office, with a fine, large baby boy, four months old, to show me the results of an operation for tuberculous bowel.

CASE IV.—She was twenty-four years of age at the time she first consulted me; had constant diarrhœa with discharge of pus and blood; had a pulmonary lesion which was now quiescent; was constantly running a fever; was exceedingly thin and pale. The abdomen was opened. Large, ulcerative, infiltrated masses were discovered along the cæcum, ascending, transverse and descending colons. The appearance was that of tuberculous bowel. In fact, it seemed to be not possible of mistake.

The ileum was drawn out through the rectus muscle and allowed to remain for about a year. She had improved but there was still a purulent discharge. Proctoscopic examination showed that there was no involvement of the sigmoid. Therefore, a second operation was performed, at which time the sigmoid was cut, the proximal end brought out through the left rectus muscle, to act as drainage for the large intestine. The proximal ileum was cut off just above the fistula and implanted into the pelvic colon, while the distal end of the sigmoid and the distal end of the ileum were turned in. Pus and blood streaked mucous continued to discharge from the open end of the sigmoid for several months. Gradually it decreased until finally after about two years, nothing but clear mucous escaped. After this had continued for several more months the ileostomy fistula was closed by first using a clamp, after the Mikulicz plan, to cut the spur and permit the mucous in the stub of the turned-in distal ileum to go on into the large bowel. Then the fistula was closed by the extra-peritoneal method shown by the pictures. She has gained until she now weighs nearly 150 pounds; has become pregnant and borne a perfectly healthy child. Of course this chronic mucous fistula is a slight annoyance, but we believe it is far safer to leave this fistula than attempt another operation for the purpose of removing the intestine. Removal of the transverse colon involves serious injury. Removal of the omentum with the transverse colon is not a simple thing. It is considered more serious from the standpoint of morbidity than mortality.

CASE V.—The next patient I exhibit here is a young woman who came to me more than two years ago, with obstructive symptoms, dysentery, discharge of pus and blood, and a mass in the pelvis. We opened the abdomen and found an enormous tuberculous mass, in separating which we opened the sigmoid, which was tuberculous; had apparently become so from an adjacent Fallopian tube which seemed to empty into the bowel. There was a very extensive area of tuberculosis of the peritoneum in the neighborhood. Instead of trying to remove the mass, which we knew would be impossible, we simply placed a quarantine around the area, brought the ileum out through the right rectus muscle, by the method shown in the pictures.

Her husband was in the army and she was being cared for by the Red Cross. She weighed less than one hundred pounds and was rapidly going down. There had been constant purulent discharge from this wound, part going through the rectum and part coming out through the side. The fistula presented typical everted edges characteristic of a tuberculous fistula. Water through the rectum or through the ileostomy wound passed out through the side. At the present time the fistula is closed part of the time but occasionally opens. The patient, when it is closed, as it is now, is very insistent upon closure of the ileostomy wound. She now weighs 140 pounds and as far as can be seen is in perfect health. We still have hopes that this tuberculous fistula will finally permanently heal from absolute rest, just as her intestine had healed.¹

CASE VI.—The next patient which is for operation was referred by Doctors Matson and Bisiallon. The patient had been sent to them for pulmonary involvement. He is eighteen and a half years of age, was refused army entrance two and one-half years ago because of his tuberculosis. Since last September the patient has had dysentery, pain

¹ On the 14th of June, 1921, the patient came back, still in perfect health. X-ray showed no particular narrowing of the lumen of the sigmoid. The fistula had been healed for more than four months without breaking open. There was no purulent discharge from the colon and no pus could be washed down by irrigating through the ileostomy wound. At the time of the sending in of this manuscript, October 1st, the patient has been perfectly well. The ileostomy was closed by the extra-peritoneal method shown in the accompanying pictures, before a clinic held during the Oregon State Medical Association meeting, June 26th. It may be stated that the spur or septum had been destroyed by clamp pressure ten days before, or June 14th, 1921.

along the ascending and transverse colon, beginning to lose weight, has temperature of about one hundred in the evening, and his condition has been diagnosed by Doctors Matson and Bisiallon as tuberculous enteritis. We have concurred in the diagnosis.

We will first make an exploratory incision large enough to admit the hand and to observe the condition of the intestines. On examination we find extensive thickening and evidence of ulceration along the transverse colon but more extensive in the cæcum and the last six inches of the ileum. You will note here very marked thickening, which is undoubtedly tuberculous enteritis. The disease is too widely disseminated to consider the question of removal. Therefore, we will bring out a loop of the ileum and sew its limbs together so that after the fistula has served its purpose we will be able to close it without having to open the abdomen. I do not know just what technic Dr. John Young Brown used in making his ileostomy wound. Owing to the fact that we have devised a method described in this connection, of closing a fecal fistula without opening the abdomen, we have adopted the plan of preparing our loop with the view of finally doing an extra-peritoneal closure.

Before closing the exploratory incision we will prepare our loop.

First, we suture the mesenteric edges together, making a sutured loop of about four inches in length. The mesenteric space back of the loop is closed with interrupted sutures, preferably No. 0 chromic catgut, which we are now using. You will note that there is no chance for the loop of bowel to work into the mesenteric pouch back of the line of sutures for we are closing it off by suturing the mesenteric sides together. We now come across the transverse diameter of the small intestine nearly to the free border and sew the free borders together with interrupted chromic sutures also. We now have two flat sides of the small intestine sutured together. In a few days we will open the apex of this loop and create our fistula. After the fistula is established, if we will pass a thumb down into one side and a finger into the other, we will find two flat walls of the intestine sutured together. (Figs. 1 to 5.)

This may, at first thought, seem useless, but it is a very important fact if we are to close the fistula by the Mikulicz plan combined with our method of closing a fistula without opening the peritoneal cavity.

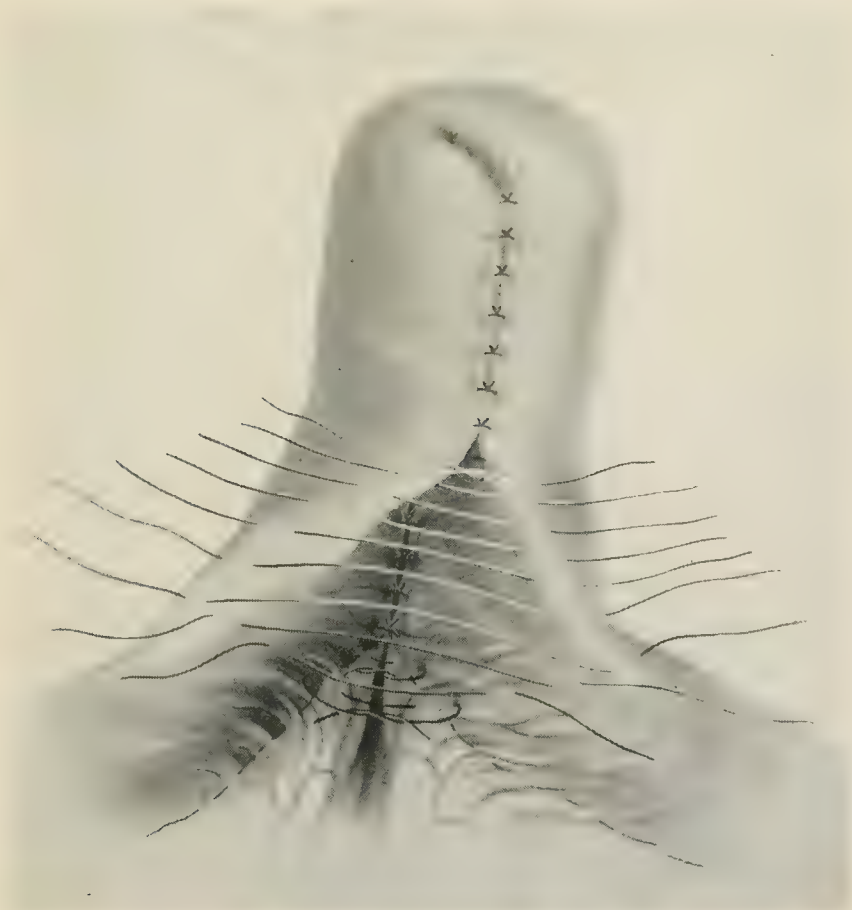
For instance, suppose we simply drew up a loop of bowel through

FIG. 1.



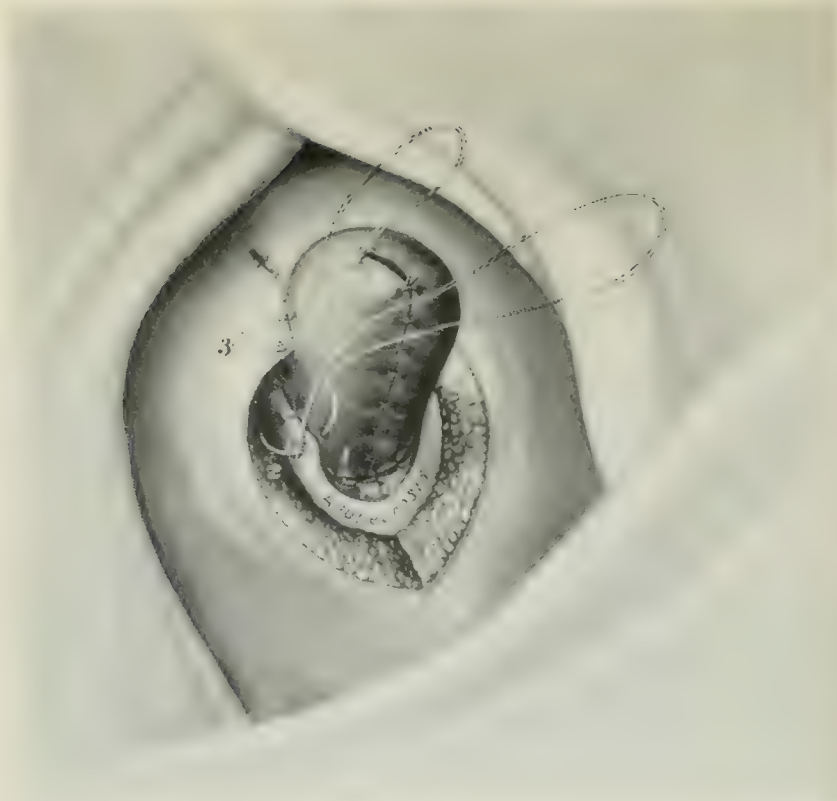
Suturing the mesenteric edges of the intestinal loop. (The suturing shown in this picture is defective in that a space is left back of the line of sutures. For correct suturing see Fig. 25.)

FIG. 2.



Suturing together the free edges of the intestinal loop.

FIG. 3.



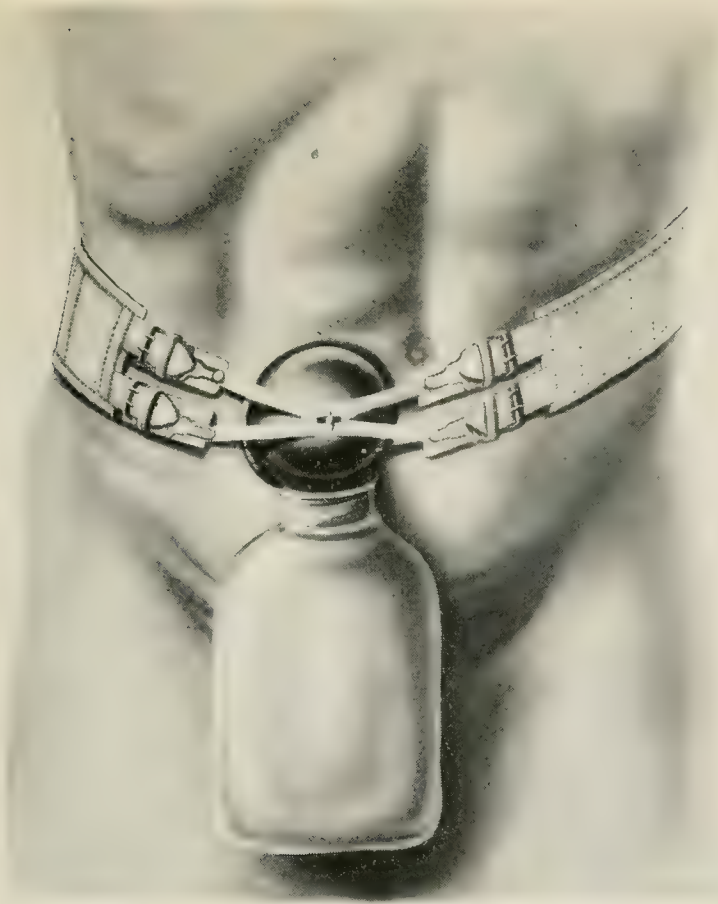
Fixation of the intestinal loop to peritoneum, aponeurosis and skin by chromic catgut doubled.
1. Peritoneal suture. 2. Aponeurotic suture. 3. Skin suture.

FIG. 4.



Sectional view of the fixed loop showing the passage of the intestinal contents through the loop.

FIG. 5.



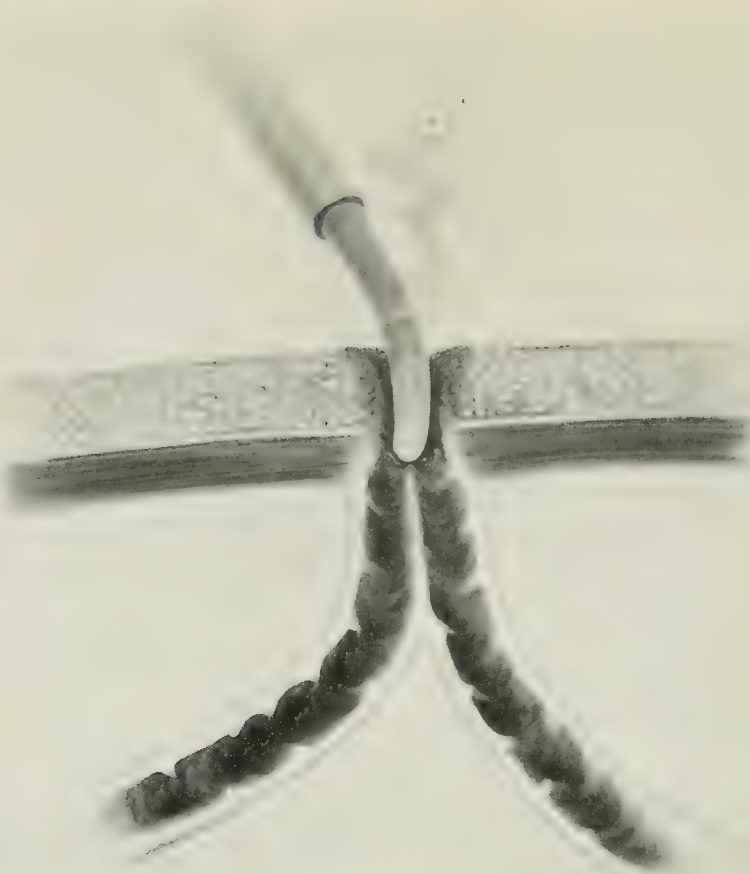
Delatour colostomy bag. (Tiemann & Co., New York.)

FIG. 6.



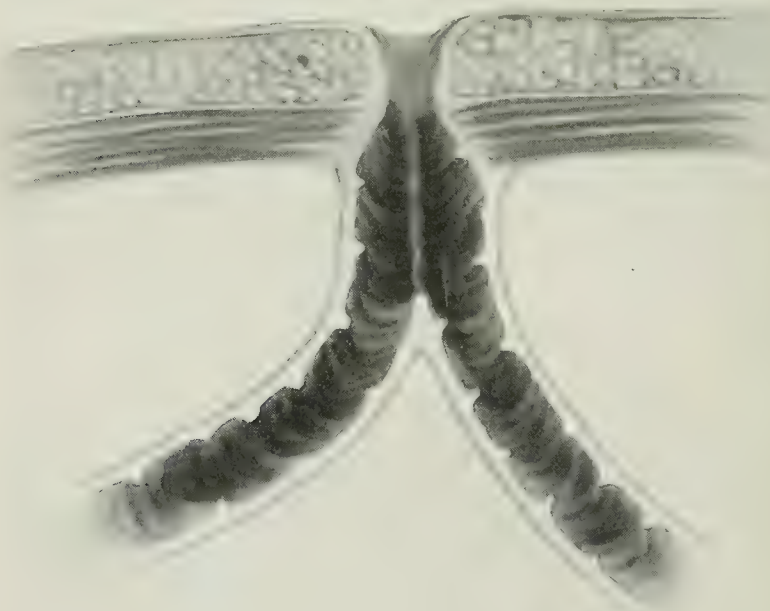
Gradual destruction of the septum between two limbs of loop with clamp.

FIG. 7.



Destruction of the intestinal mucous membrane which passes through the intestinal wall, by use of the cautery.

FIG. 8.



Intestinal fistula after the cauterized area has healed.

a hole and fastened it in the wound. The fistula would work all right, and if we intended to open the abdomen afterward it would be just as good as any other fistula, but in this case we hope to be able to close the fistula without opening the abdomen. Therefore, we will have to crush this septum by a pair of forceps.

Suppose we had not sewed this loop together. When we put in our clamp later on to cut the septum a high loop of jejunum might be fastened in the angle of this loop. It would be caught in the forceps. We would thereby create a jejunal fistula, which next to a duodenal fistula, is one of the most serious accidents which can happen to us in abdominal surgery. When we get a jejunal fistula we usually wait, hoping that the patient will get better and that the fistula is not so serious as we think. Finally the patient grows weaker until he becomes too weak to stand an operation. The result is a very large mortality in high jejunal fistulæ as well as in duodenal fistulæ. In sewing the loop together in this manner we avoid any possibility of such an accident. We can cut the septum between these two limbs with impunity, as a preliminary step to closing the fistula.

Having thus prepared our loop, we now make a stab wound through the right rectus, one and one-half inches to the right and an equal distance below the umbilicus. We pick up the peritoneum with forceps on each side, draw the loop up through the hole. We now take this double chromic catgut, No. 00, and attach this loop all around to the parietal peritoneum by a lock-stitch. We use a lock-stitch to prevent constriction by a purse-string effect of a continuous suture and at the same time avoid the tediousness of interrupted sutures all the way around.

Now that we have completely surrounded the loop we pick up the aponeurosis with the same thread and sew it around the loop with the same kind of lock-stitch. Finally we take the same suture and thread it into a cutting needle. Here we will sew the skin to the loop of bowel with the same kind of sutures.

Sometimes we slip a tube underneath the loop. Ordinarily that is not necessary in an ileostomy. The tendency to draw in is not so marked in the small intestine as in the large.

It is quite essential not to make this too tight around the loop, otherwise, the patient may begin to vomit and it may be quite serious, for the normal swelling following the operation increases the tightness.

If the patient should begin to vomit and show evidence of intestinal obstruction, to-morrow or next day we will open the bowel.

We will put the young man under good sanitary conditions and if he recovers entirely and in the course of a year or two becomes perfectly healthy, we will close the fistula.

In practically all the cases we have had the patient begins to urge the surgeon to close the fistula within a few months or a year. I have usually made it a point to put the patient off for at least another year before finally closing it. So far we have not had to re-open the abdomen, if we have made this extraordinary delay. Some of the reported cases of treatment of ulcerative colitis by the Brown method have not been so satisfactory. But I have noted that in these cases reported not cured the fistula was closed by the surgeon within six months, or at most a little more than a year after the ileostomy was performed. Comparing these results with our own, in which we have left the fistula from one and one-half to two years after the patient thought herself entirely well, and from two to three years after the ileostomy opening, the lesson seems to be that the long time opening is far the safer plan, although it is quite annoying to the patient.

I wish at this time to exhibit another case of tuberculous bowel, which was likewise referred by Doctors Matson and Bisiallon.

CASE VII.—You will note that there is a small fistula through the left rectus muscle, which shows the definite ear-marks of tuberculous fistula. Note the everted very red edge which looks almost like mucous membrane, but which is not. This patient came three months ago, having been recently operated on for tuberculous peritonitis.

The patient was in a low state of health, very anemic, high fever and very thin. During my absence in the east Doctor Holden was kind enough to look after my work, and he advised the patient against operation and gave a very bad prognosis. When I returned I gave an equally bad prognosis, but from my former experience I still had a faint hope that the fistula might fortunately be his salvation. This hope was well founded for the man who had been in an apparently hopeless condition has gradually improved in health. The tuberculous condition has evidently greatly improved. The bowel contents are quite normal. He is feeling well and has gained a good many pounds in weight. His blood has improved and he is decidedly better. I exhibit this case for the purpose of

demonstrating the fact that if tuberculosis is present, we may be too much afraid of a tuberculous fistula. If a tuberculous fistula develops from a tuberculous process in the lower ileum and this fistula becomes complete, then we may use it for the same purpose as an ileostomy. I am inclined to think that is what happened to this young man. A tuberculous fistula has developed, which has relieved the diseased part of the intestine from its duty for a few months. It is entirely possible that this fistula has closed too soon, and it is quite probable that had we been able to keep it open it would have been much better for the patient, although he is in an improved condition at this time.²

I think the most interesting clinic we saw during the meeting of the Clinical Congress of Surgeons in Montreal, in October, was that of Doctor Archibald, who, as you may know, is doing probably the best work in surgery of the tuberculous bowel that is being done. He showed a number of most interesting cases and his conclusions from his large experience are very much the same as mine have been from my smaller experience.

One very interesting statement that he made during the meeting there was that a patient did not seem to do well if more than three or four feet of the lower ileum had to be sacrificed by short-circuiting or removal operation for tuberculous bowel. This is very interesting to me and I must confess I am not quite able to understand it. I wonder just what may be the cause of it as this is not true in other conditions. I think probably that in ileostomy even this high up would not be serious. Of course if tuberculosis is entirely localized and we are sure there are no ulcerations lower down, it is possible to remove a section of the intestine, particularly if the cæcum is the part involved.

A very interesting statement of Doctor Archibald corresponds with two or three cases of mine. It was that complete rest of tuberculous large intestine will often produce a very marked improvement or cure of pulmonary tuberculosis. He has operated on a good many cases of pulmonary tuberculosis who have enteritis with it, with very startlingly good results. I am inclined to think that this is

²This patient remained under treatment till Sept. (6 months later). In the meantime the fistula had practically healed and the bowels were in good condition.

the class of cases in which Lane has reported cure of pulmonary tuberculosis by short-circuiting operations.

Thus in discussing either inflammatory or simple ulcerative processes and tuberculosis, the same old principle of rest applies.

Many decades ago Hilton wrote his famous book on Rest and Pain. Probably no greater single contribution was ever made.

We find that in most of the ulcerative conditions of the colon, tuberculosis here, as elsewhere, is cured chiefly by rest. Rest is practically the only method of treatment of tuberculosis of the hip or knee. Lung specialists are now depending very largely upon absolute rest of the lungs by collapsing the lungs, as J. B. Murphy and others recommended more than twenty-five years ago. Therefore, the fundamental principles of an acute ulcerative condition, or tuberculous condition of the large bowel is rest. Rest is applicable in a much larger number of cases than radical surgery and is probably more reliable in the long run.

To produce rest for the cure of this class of cases of intestinal disease there are three steps:

First, making an ileostomy by the method we have just described in order that it may be closed without doing a major operation. If the disease has been extensive an ileo-sigmoidostomy should be done, after all evidence of disease has gone—leaving the fistula still open as a precaution.

Second, after the fistula has served its full purpose the septum carefully made at the first operation is cut by placing a clamp on it, with one blade in the proximal and one in the distal limb of the loop. Partially close the clamp so as to make moderate pressure. The next day close the clamp still tighter and the third day still tighter. After about three days of this pressure, if the clamp is strong, the septum, which is included in the forceps, has been killed and will very shortly cut through. This is known as the Mikulicz operation, which I shall refer to later in another class of cases. If the disease has been milder the ileo-sigmoidostomy will not be necessary.

Third, closure of the fistula by the following method:

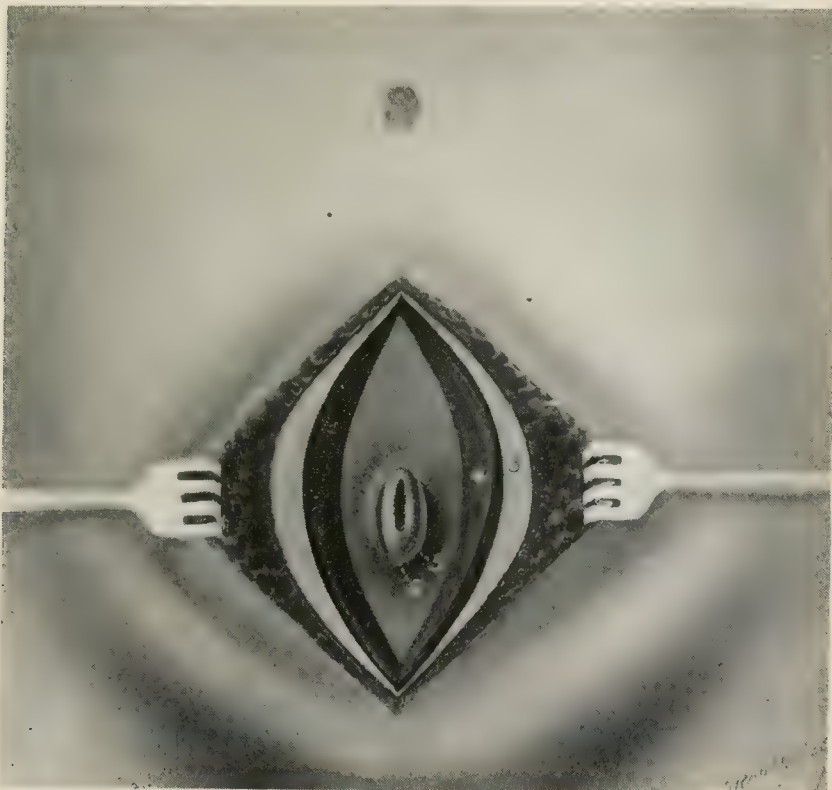
1. Dissect out the old scar down to the fat, and make an incision around the fistulous tract, including a small strip of skin, direct the point of the knife slightly away from the fistula so that it first comes in contact with the fascia about half an inch away from the fistula,

FIG. 9.



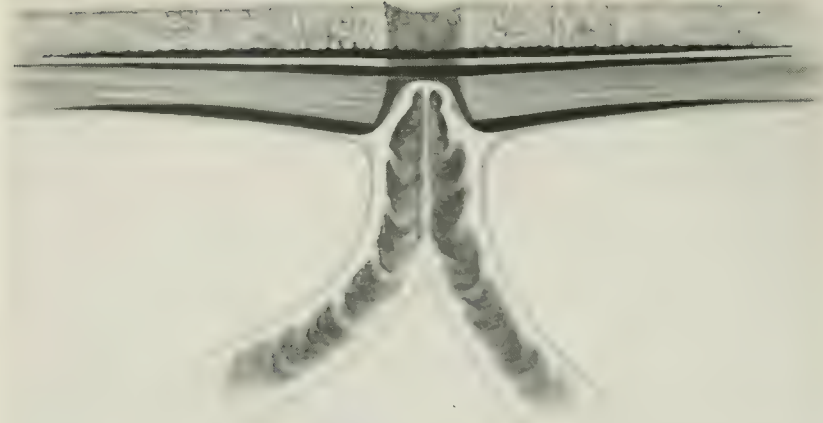
Incision for freeing the fistula from the abdominal wall and separating the layers of the abdominal wall.

FIG. 10.



Fistula freed, and layers of the abdominal wall separated ready for closing.

FIG. 11.



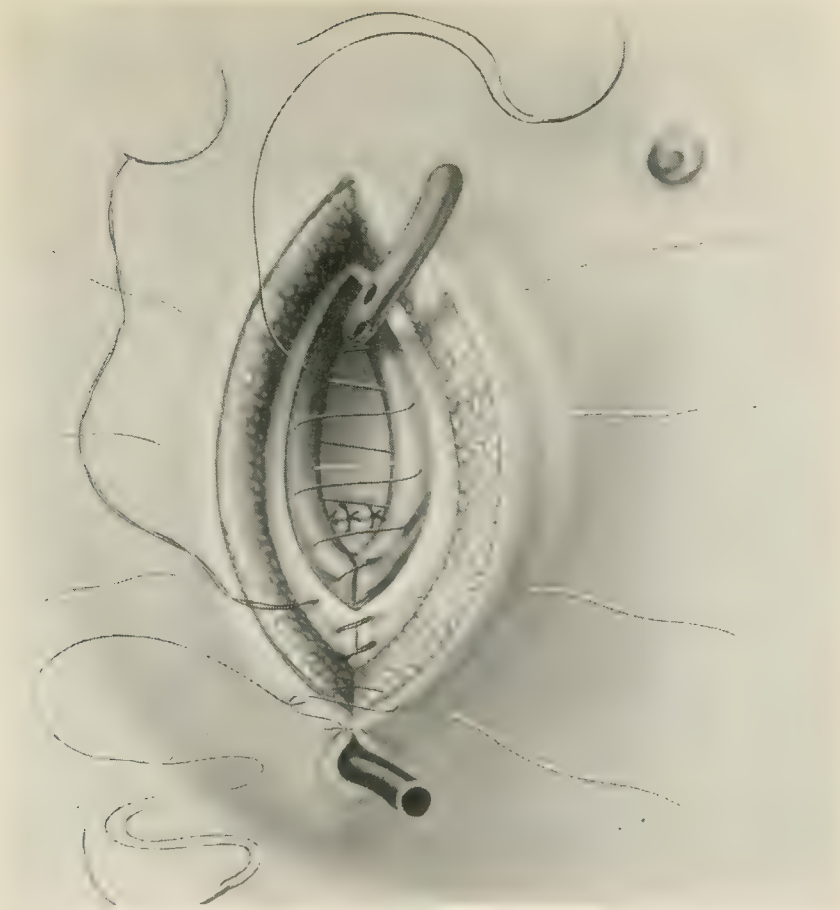
Sectional view of the fistula freed, and the layers of abdominal wall separated ready for closing.

FIG. 12.



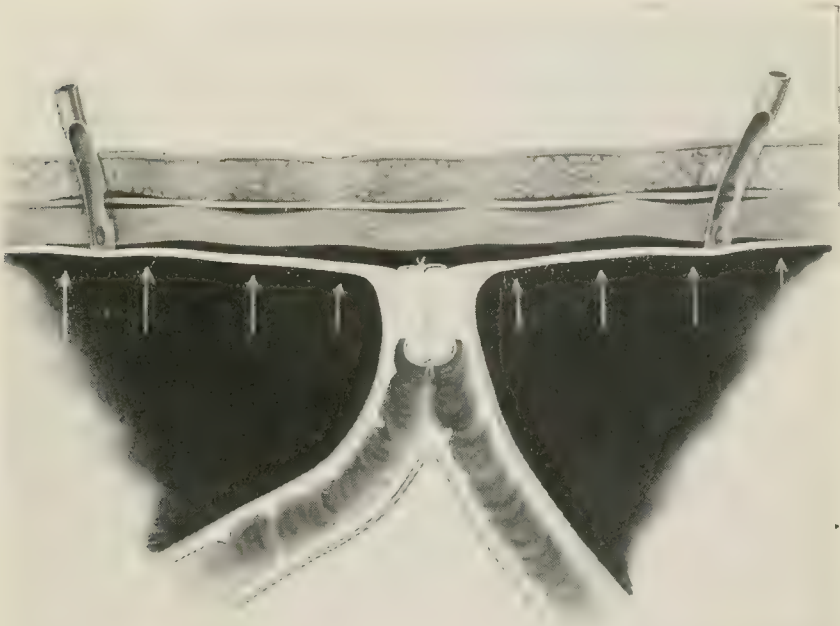
Closure of the intestinal fistula.

FIG. 13.



Closure of the abdominal wall, showing the closed fistula in the bottom of the wound and drainage tubes at the ends.

FIG. 14.



Longitudinal section of the completed operation, showing how the spaces drain around the ends of the tubes.

in order to avoid any possibility of opening the peritoneum. The incision should extend two inches or more above and below the fistula to allow for wide dissection.

2. Dissect up to the fat from the fascia for as much as two inches from the incision, draw it back, and clean off the fascia.

3. Make an incision through the fascia, beginning at the upper end of the wound and coming toward the fistula. Dissect the fascia from the muscle for at least two inches in every direction.

4. Dissect the muscle from the peritoneum in the same manner so that the peritoneum hangs loosely with the fistula standing up in its center, like a volcano and its crater.

5. The little margin of skin which has been left with the edge of the fistula is now trimmed off.

6. If the wall of the fistula is hard and cicatricial, making it difficult to turn it in, it is well to make an incision part of the way through the cicatricial tissue so that it may be turned in easily.

7. The edges of the intestine are turned in with linen sutures which are knotted on the inside.

8. A second layer of sutures brings the edges of this incision and the connective tissue over the peritoneum along with the scar tissue, covering the turned-in fistula, to add temporary strength and bulk to the closure. The peritoneum and the rest of the wound is now thoroughly mopped or irrigated with salt solution to make it as clean as possible.

9. Silkworm sutures are passed through the skin, fat, fascia, and muscle about a half inch or more from the edge, and are left untied, space being left at the upper end of the wound for drainage.

10. Suture the muscle loosely with a continuous catgut.

11. Suture the aponeurosis with a strong double catgut.

12. Suture the skin with a horsehair button-hole stitch.

13. Place the drains, which may be tubes or cigarette drains at each end of the wound. (See Figs. 5 to 15.)

The most fundamental principle connected with this method for the closure of a fecal fistula is drainage. The principal point necessary for producing this drainage is wide dissection of the various layers from the wound so that the infected material has an easy passage way to the drains placed at the two ends.

It will be seen by the pictures that drainage is in all directions away from the fistula, which has been sewed.

We have never failed to close a fistula of either the large or small intestine by this means when no obstruction existed below, provided there was not sufficient scar tissue from former operations to prevent separation of the abdominal wall into its layers.

The old method of closing a fecal fistula by simply freshening its edges and sewing it was practically always a failure.

By this stage-operation or modification, we make of John Young Brown's ileostomy an operation practically devoid of danger.

There is one class of cases in which rest does not play such an important part, namely, cancer.

CANCER OF THE RECTUM AND SIGMOID

Of course, we know, and have known for a long time that a colostomy will greatly prolong the patient's life, who has a cancer of the rectum, although the cancer is not obstructive. In some such cases of colostomy, the patient has lived as long as five years after the operation.

I have observed a very interesting thing in connection with two-stage operations for carcinoma of the stomach. It is that when we open up for the removal of the pyloric end of the stomach, after we have done a preliminary gastroenterostomy, we invariably find the growth diminished in size. In other words, there is an infection taking place in the neighborhood of the ulcerative cancer, which apparently is playing an important part in the general welfare of the patient. A short-circuiting operation produces rest and rest retards the activity in the neighborhood of the growth. This probably explains the prolongation of the life of the patient by short-circuiting operations in cases of carcinoma even when there is no obstruction. Particularly is this true in cancer of the rectum.

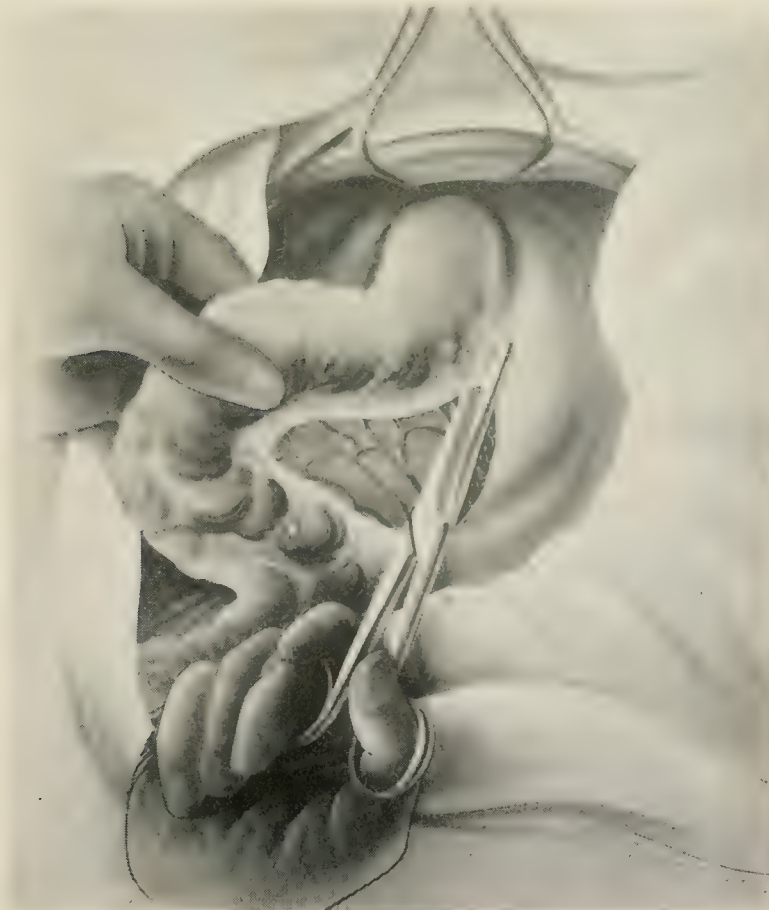
In the *Annals of Surgery*, April, 1915, an article of mine was published on the "Major Procedure First in the Two-stage Operation for Cancer of the Rectum." In this article attention was called to the fact that the statistics of the Mayo Clinic had suddenly been cut from a mortality of 25 per cent. to a mortality of 12 per cent. by doing the operation in two stages. The members of this clinic had used several different methods of application of the two-stage operation. In one, a very simple colostomy was performed without

FIG. 15.



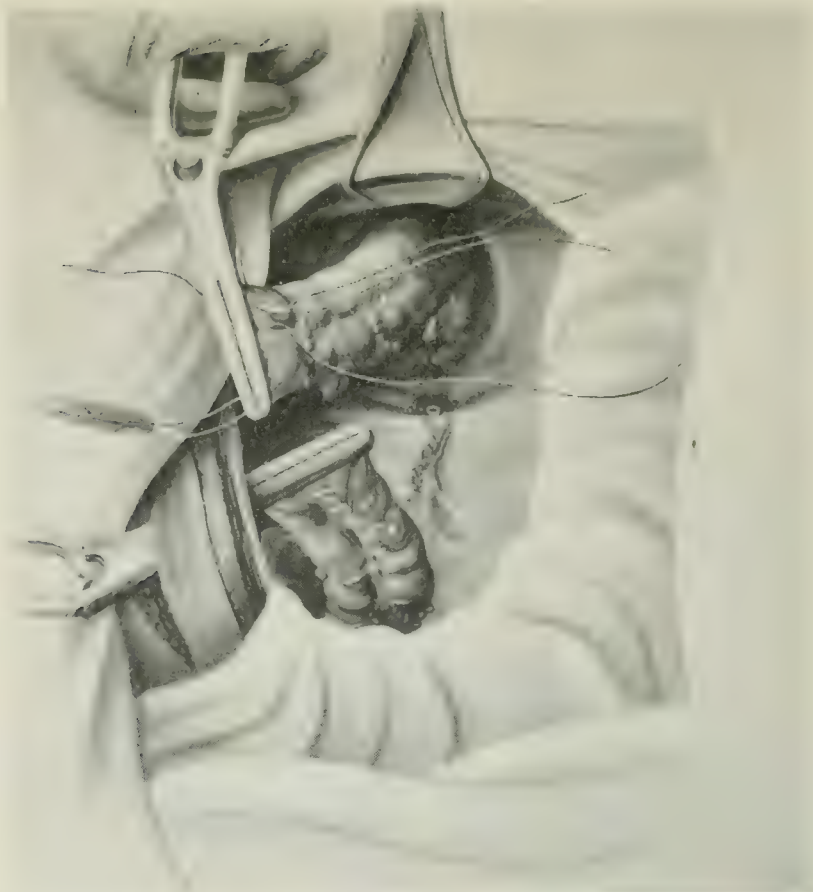
Cross-section showing how all the lines of sutures are enclosed in the silk-worm sutures, which obliterate spaces and allow drainage to either side between the layers.

FIG. 16.



The sigmoid is mobilized by cutting the peritoneum on each side of its mesentery.

FIG. 17.



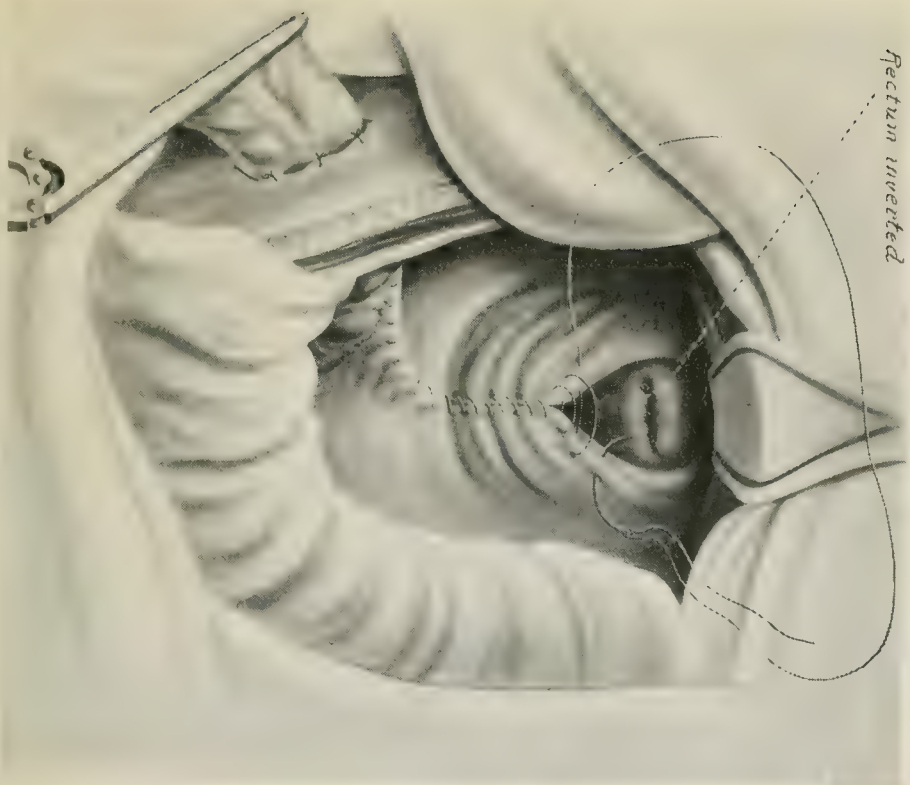
Clamping and cutting the sigmoid after the vessels have been ligated. Note that one of the clamps passes through the stab wound in the left rectus muscle.

FIG. 18.



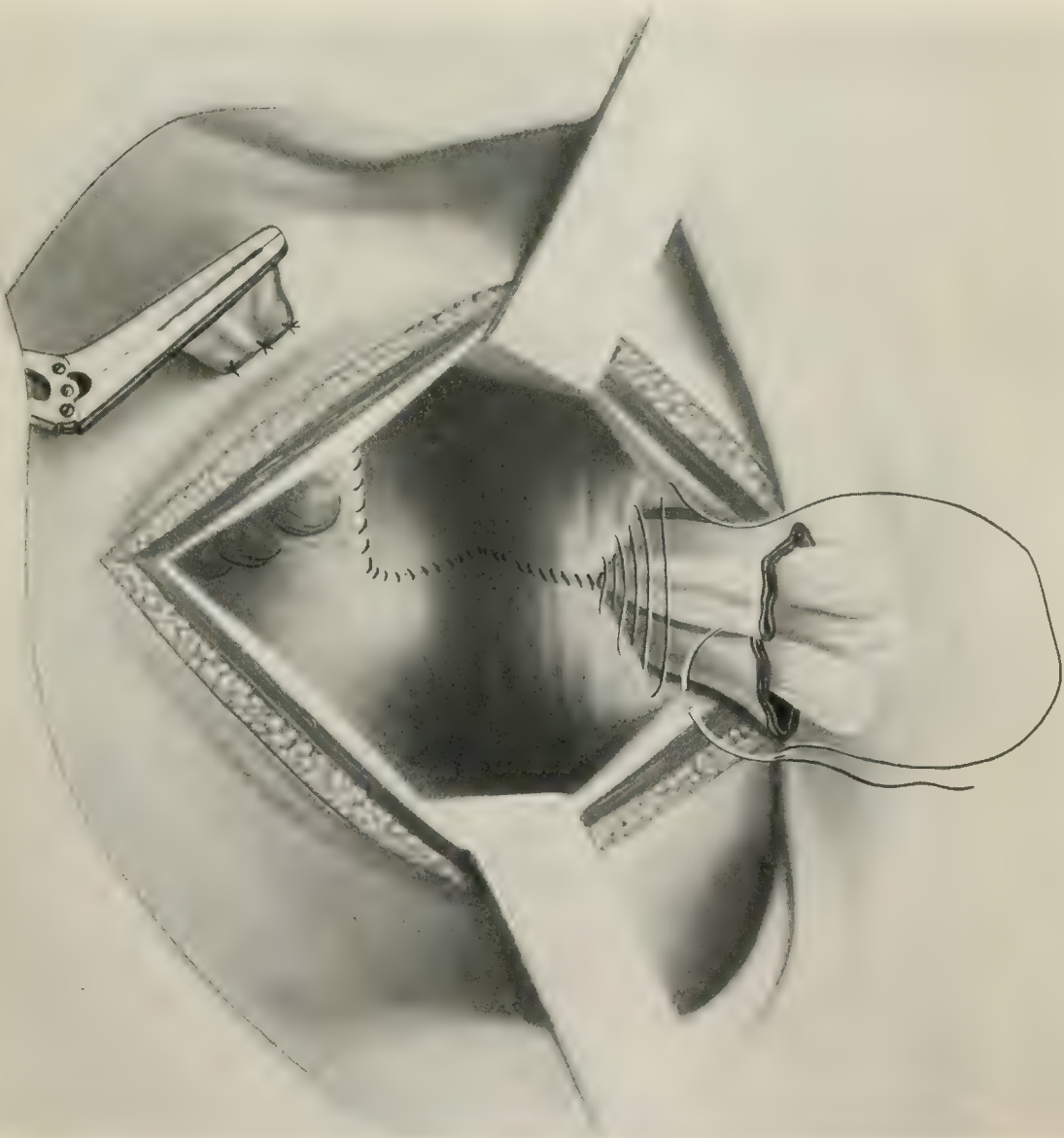
Proximal sigmoid held in clamp. Tube is passed up to end of distal sigmoid, where it is fastened by a strong double suture passed through the intestine and eyes of tube and tied. When the intestine is held by two forceps and traction is made on the tube, inversion is produced. Note the ends of the severed superior hemorrhoidal artery.

Fig. 19.



After the distal sigmoid has been inverted and drawn out through the anus, the inverted end is closed by three or four interrupted catgut sutures, and a continuous catgut suture is run along the mesosigmoid, covering the raw fat edges with peritoneum from the proximal sigmoid to the bottom of the cul-de-sac.

Fig. 20.



In man a drain is inserted and fixed by catgut sutures to the turned-in end of the rectum. The suture line, shown in Fig. 19, is continued, drawing the peritoneum of the pelvic wall around the drain, completely closing off the peritoneal cavity from the drain.

FIG. 21.

*Sectional view of Peritoneal
Fold surrounding Drain*

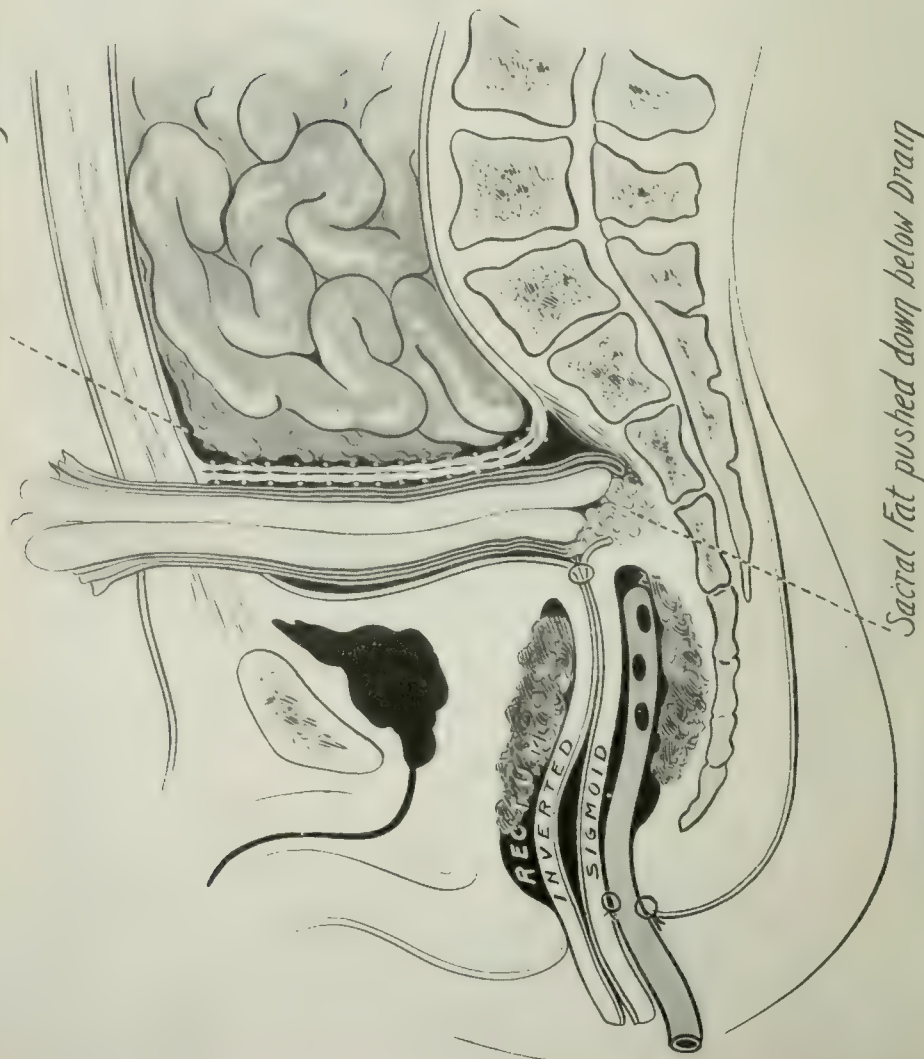


FIG. 22.

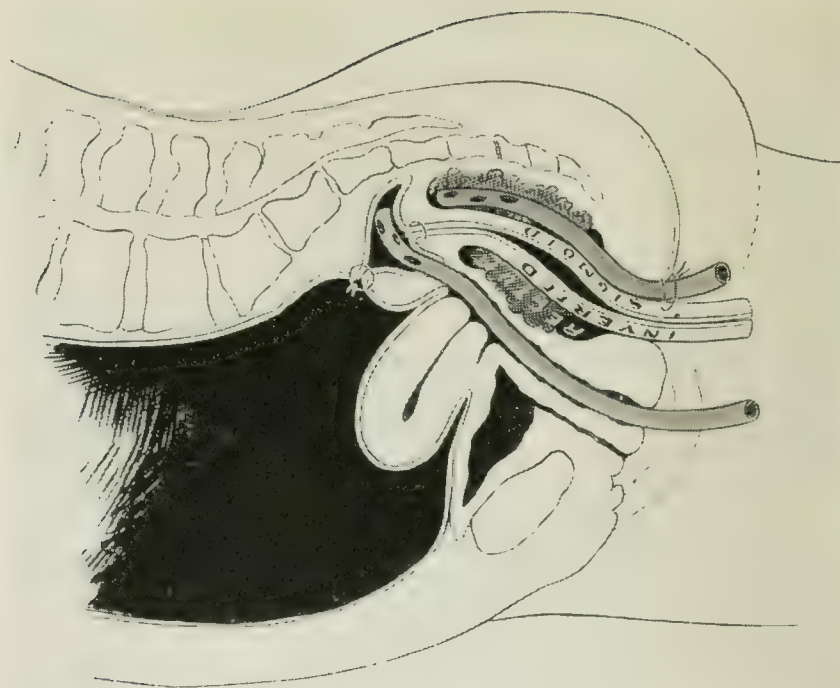


Diagram showing the supplementary drain passed through a stab-wound in the cul-de-sac in case of woman.

Sectional view of drains which have been tubularized by the peritoneum of the pelvic wall in man.

severing the intestine at the first sitting; in another, the intestine was severed, the end turned in, the proximal end brought out through the abdominal wall, and the distal end dropped down into the pelvis at the first sitting. They recorded one or two cases of perforation of this segment, however, which caused them to abandon it. C. H. Mayo has recently improved this by closing the peritoneum over the stump, and doing the second, or removal, operation five or six days after the first stage, before sloughing and abscess-formation takes place.

The point we brought out was that the superior hemorrhoidal artery was ligated just below the promontory of the sacrum, a rectal tube was passed up by the growth, the end of the closed distal segment fastened to the end of the rectal tube, the tube withdrawn, and with it the distal end of the sigmoid colon was drawn through the anus. The invaginated portion was then closed over all, making a complete abdominal operation, the fat and glands in the hollow of the sacrum having been pushed down with the rectum. (Figs. 16 to 22.)

Eight cases were reported in that article with one death. Since that time I have performed operations on twenty-two other patients by this method, with some modifications and have had no further deaths, making in all, proctectomies on thirty patients with only one death.

There was a serious objection to this original operation, namely, the tying of the superior hemorrhoidal artery deprived some of the pelvic tissues of nutrition to such an extent that there was a good deal of sloughing, including the upper portion of the rectum, the pre-sacral fat and other tissues, so that the patient sometimes had a very stormy week or two, running a temperature of 102° or more. Finally the infected area would break into the rectum and discharge. This feature was serious only in one case, and that was one in which a septic phlebitis of the veins of the left leg took place, thereby delaying recovery for some weeks. I may incidently state that this patient is still alive, seven years after the operation. I have always had an idea that a certain amount of sloughing and infection following the removal of a cancer was probably beneficial for final results.

This feature was very annoying so we soon developed means of avoiding it. Drainage, of course, was the solution of the question.

Drainage of the devitalized area around the end of the inverted rectum was easy in women by making a stab wound through the

upper end of the vagina into the cul-de-sac. In men, however, the problem was more important, owing to the fact that men are more prone to cancer of the rectum. We soon found, however, that the narrow pelvis of a man lent itself particularly well to the drainage proposition. First we simply passed a large cigarette drain down to the end of the rectum and brought it up through the free peritoneal cavity through the lower end of the main exploratory abdominal incision through which we operated. This defeated our original ideal in that we still had an open peritoneal cavity. We soon found that the peritoneum of the male pelvis could easily be drawn around the drain. Continuing the suture which was described in the original operation, the peritoneal suture is continued forward, drawing in the peritoneum of the lateral wall of the pelvis above the drain until the front abdominal incision is reached.

In this way the drain is left in a peritoneal tube. There is no foreign body and no raw surface in the free peritoneal cavity.

CASE VIII.—I have here a patient who had this first operation performed about twelve days ago. You will note that the large cigarette drain with the large safety pin through it is still in the lower angle of the wound. We will allow this drain to remain as a guide to the operation we are going to do to-day, and until three or four days after removal of the rectum. You will observe that the incision through which this comes, is a long incision and has been made through the right rectus muscle, an inch or more to the right of the median line. This long incision we make so that we can examine the growth more carefully and also can determine whether or not there is an extension of the growth to other organs, which would make it useless to do a radical removal operation. Through this right-sided incision we perform the technic of most of the operation. The exploratory incision is made through the right rectus because through the left we expect to bring up the colon for colostomy. By having this large incision the operation is easy, and at the same time we are able to make our stab wound in the left rectus wound just the size we desire, for bringing out the single gut, as shown in the picture.

Note the small rosette of everted mucous membrane, compared with a colostomy in which both limbs of the loop have been drawn up.

We will now put a large pad of gauze and cotton in front of the abdomen and turn the patient over on the face. This table, as you will note, breaks in the middle. We have put the break just at the point where the hip joint bends. We make our incision slightly to one side of the sacrum and coccyx, down the perineum and around the anus, catching the bleeding vessels. We now expose the sacrum and coccyx, and with a hand chisel separate the last joint of the sacrum from the fourth, remove the last joint of the sacrum and all the coccyx. Just in front of the sacrum is a small bleeding vessel.

We now cut the muscle around the rectum so as to make room. We feel the growth in the front wall of the rectum. We have no fear of hemorrhage. We are able to control the branches of the pudic artery, which supplies the perineum, with forceps. All the higher blood supply has been destroyed by our ligature at the first operation. I now insinuate the fingers of my left hand between the sacrum and rectum. You will note that there is no hemorrhage taking place. The muscle and fascia at the side, we grasp in forceps and cut, but there is no hemorrhage.

I now push the tips of my fingers farther up until I touch the large cigarette drain which you saw on the abdominal side, and through which was a large safety pin so that I will not be able to draw this drain by inadvertant pulling. The drain, I know, marks the upper part of the tissues to be removed. I saw to that at the first operation. I know that this drain is from one to two inches below the normal peritoneum.

You will note how the œdema following the other operation apparently makes it easier to separate the fat and rectum from the pelvic wall. My finger tips are entirely above the end of the rectum, which was turned in and is not opened. I will now gradually pull the inverted rectum with any remaining fat downwards and separate it from the base of the bladder. This growth is quite extensive and you will see here that I have accidentally opened the rectum and have broken into the growth. I do not fear this because I am going to leave the drain anyway. I think the drainage will make grafting of the growth into the tissues very unlikely. Now we cut all the muscle and remove the rectum entirely with the anus. Now the muscles of the perineum are sutured with catgut and the skin with horse-hair. We leave a large cigarette drain in the wound rather

loosely placed. The patient will be required to lie on his back a portion of the time to permit of gravity drainage.

We will expect this wound to heal by primary union except where the drain comes out. In three days we will remove the upper drain and leave the main part of drainage for the action of the drain we are now placing.

The advantages of this technic are several.

First, the primary operation is a clean operation, at which most of the finer points of technic are performed.

Second, the main blood supply of the rectum and fat tissues, including the lymphatic glands in the hollow of the sacrum is destroyed by ligation of the superior hemorrhoidal artery. It is almost certain that with the destruction of the vitality of the tissue in the hollow of the sacrum goes the vitality of the cancer cells. It is, therefore, reasonable to believe that the ligation of this artery removes much tissue and possibly many cancer cells by starvation, which would be very difficult to do by dissecting surgery.

Third, the peritoneum having been closed at the former operation, the main blood supply having been destroyed, the removal of the rectum becomes practically a minor operation.

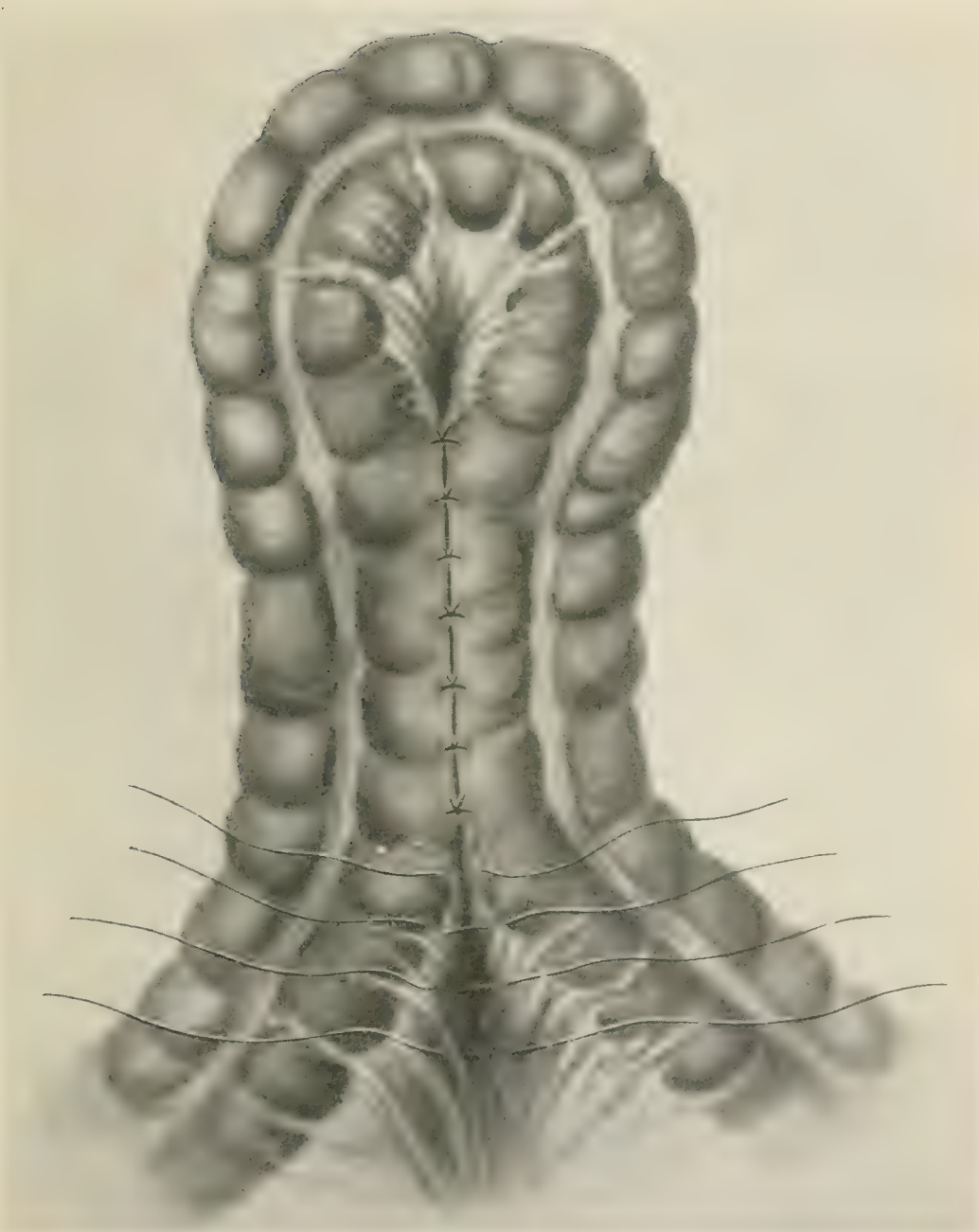
It is needless to call your attention to the fact that this operation, of course, is applicable in only a limited number of cases. The growth must be well below the peritoneal folds and in the rectum proper. There must be a sufficient opening to pass a good-sized rectal tube with ease, so that the inverted sigmoid may be readily drawn down through the anus. The technic of the operation you will see in the pictures more clearly than I can show you in doing the operation itself. (For steps in technic see Figs. 16 to 22.)

If the growth is too extensive to be removed with hope of a fair degree of success, we are now attempting to treat it with radium, after doing a colostomy.

CASE IX.—Our next patient here is one on which we have done a colostomy as a preliminary step to using radium.

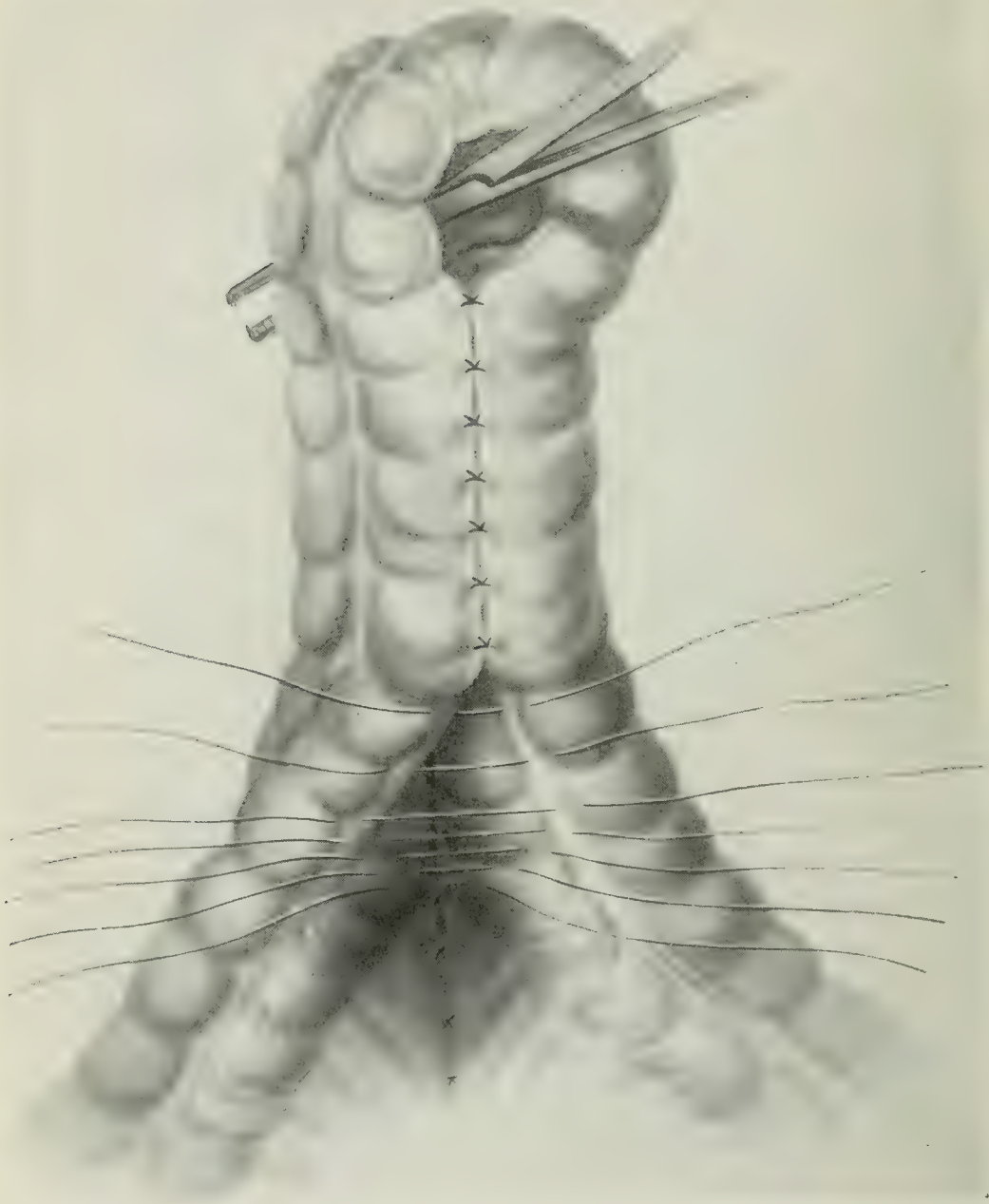
You have no doubt noted the difficulty, in certain cases, of keeping a colostomy open. The tendency of the large intestine is to draw back into the abdomen unless it is fixed in some way. The methods of permanently fixing the colon in the wound described by Mixter and Sistrunk, which include drawing various layers of the abdominal

FIG. 23.



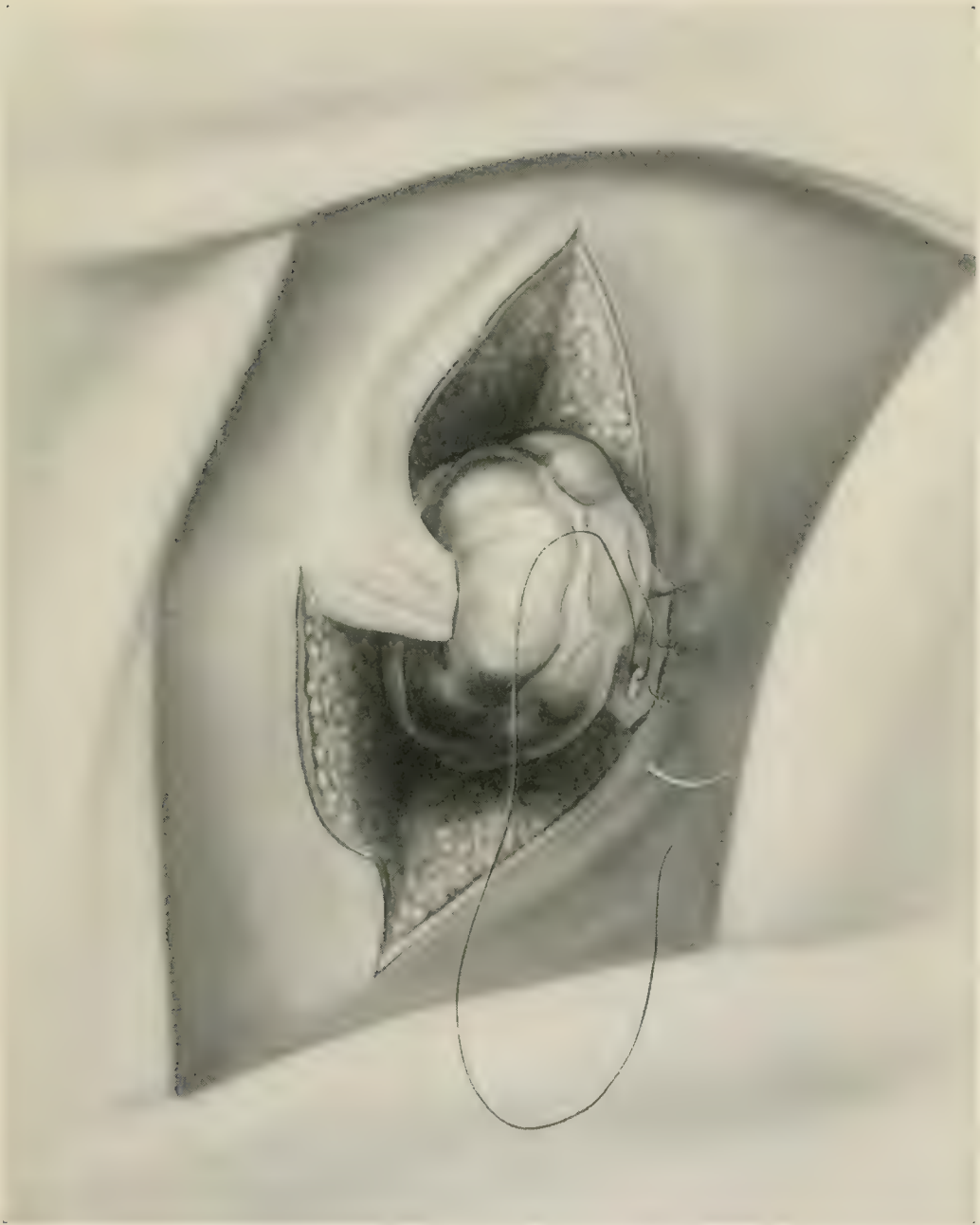
Correct method of suturing mesenteric edges of the intestine as first step in preparation of the loop for temporary enterostomy or colostomy.

FIG. 24.



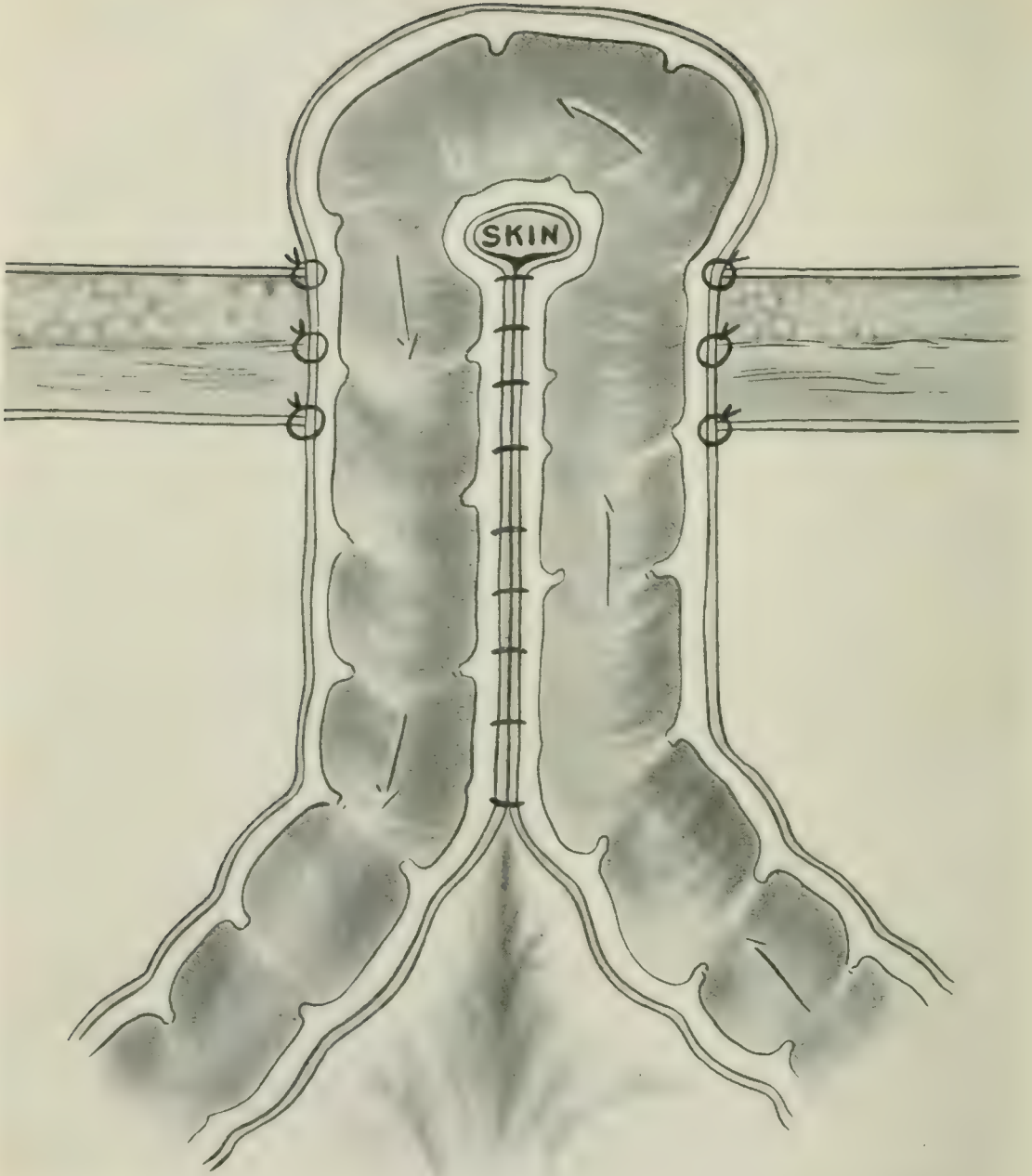
Suturing the free edges of longitudinal bands for the purpose of producing safe surfaces for later destruction of the septum. Forceps passes beneath the intestine at the apex of the loop for the purpose of drawing through the flap of skin.

FIG. 25.



The loop has been sutured to the peritoneum and aponeurosis by rows of fine double chromic catgut used as a lock-stitch. The skin flap has been drawn through the loop and is being sutured to the skin on the opposite side by chromic catgut sutures. The rest of the skin wound will then be closed by interrupted chromic sutures around the loop.

FIG. 26.



Sectional view of loop showing strip of skin passing it through the apex of the mesentery. When it becomes necessary to close the colostomy the clamp placed as in Fig. 6, destroys the supporting skin strip as well as the intestinal septum.

wall through the loop of the sigmoid, forming the colostomy, are very helpful if the colostomy is to be permanent. In such cases as the one we have here, it is barely possible that we will be able to cure this cancer with radium and it is even possible that the lumen of the bowel may be restored. In such a case it will be highly important for us to have a colostomy opening which can be closed without having to open the abdomen again.

If we simply prepare a loop as we did in the ileostomy operation, shown in one of the previous cases, and sew it into the wound in the same way, it is very likely to pull into the abdomen and have a tendency to close. To meet these objections we have devised a kind of hybrid operation which we described in the *Annals of Surgery*, March, 1920, under the title, "A Permanent Colostomy or Enterostomy which may be Closed by an Extraperitoneal Operation."

In this operation the two loops of colon are sewed together. The peritoneum is sewed to the prepared loop in the same manner, also the aponeurosis and muscles: When we reach the skin we mobilize a strip of skin one-half to three-quarters of an inch in width at one end, puncture the mesosigmoid near the apex of the loop above the point where the two limbs have been sewed together, draw the detached end of mobilized strip of skin through the hole in the mesentery beneath the intestine, and attach it with interrupted chromic catgut sutures to the skin on the opposite side. Now the skin is closed around the loop in the usual manner. Figs. 23 to 26. If we are fortunate enough to succeed in curing this cancer by radium we can then cut the septum, including a strip of skin, with a pair of clamps by the Mikulicz method and close the fistula by the extraperitoneal method, shown in Figs. 9 to 15.

There is another type of case to which I wish to call attention, but of which I have no illustration to-day.

It is the *annular carcinoma of the sigmoid*, in which the growth has gradually contracted and finally presents the condition of complete intestinal obstruction. Such a patient is often in a serious condition. To attempt to do a radical removal is very dangerous for the patient. To do a simple colostomy, which is frequently done, and which we all have done before we learned of the Mikulicz operation, subjects the patient to another very serious major operation for the removal of the growth itself, plus closure of the fistula,

which in this case must be done by an intra-abdominal operation. It was for this class of cases that Mikulicz brought forth his epoch-making operation which consisted of bringing the growth with a loop of bowel, out through the abdominal wound, fastening it to the peritoneum and waiting a few hours or a day or two when the intestine proximal to the growth was opened with a Paquelin cautery without anæsthetic. The operation is without shock; is done in a very few moments, and when the intestine is opened, gives complete relief. A few days later, with the Paquelin cautery, the growth is removed and the two barrels of intestine are left in the wound to be closed later by clamping the septum and cutting the two limbs together.

Frequently a colostomy thus made will close itself without further operation. If not, it is very easy to close it by plastic operation, such as we have described. I am quite sure that this is the greatest single life-saving technical procedure that has been given to intestinal surgery. It is applicable in a great many cases.

CASE X.—I have one more patient to show you. This man came to Doctor Sears suffering from intestinal obstruction, which proved to be carcinoma, early last spring. On May 20, last, we operated for carcinoma of the left half of the transverse colon. At that time most of the transverse colon was removed, practically all the omentum, most of the transverse mesocolon, including the mesenteric glands. The appendix was removed and the cæcum anastomosed with the sigmoid colon. The proximal cut end of the transverse colon was turned in by a purse-string and allowed to remain.

The patient always had trouble afterwards, although the contents of the cæcum were delivered into the sigmoid. The condition grew gradually worse. The patient complained, giving symptoms of intestinal obstruction, although the bowels would move. An enlargement gradually developed on the right side of the abdomen with visible peristaltic waves. These waves moved from below upward. Our diagnosis was that a loop of small intestine had probably become strangulated and was partially obstructed, inasmuch as an opaque enema showed by the X-ray that the cæcosigmoidostomy opening was good.

On opening we found there was no obstruction of the small intestine; that there was a tremendously dilated ascending colon, up to the first part of the transverse colon, which had been turned in. At first it was quite puzzling to know why all these symptoms. Finally we concluded that the intestine had probably been dilated by peristaltic waves trying to force the contents of the cæcum upward in its normal wave against the turned in end of the colon. We, therefore, decided to remove the ascending colon and the first part of the transverse colon, which had been left at the former operation.

This we did, removing the ascending colon down almost to the ileocæcal valve, leaving just enough room for it to empty. We had a large cæcosigmoid opening, therefore, we felt we were all right.

This second operation was done on January 22nd, just a little more than a month ago. The obstructive symptoms have been entirely relieved and we feel our deductions were correct. In leaving the large dead end above the ileocæcal valve at the first operation and expecting all the contents of the ascending colon to be delivered backward into the sigmoid, we had made a mistake. The peristaltic waves still continued to act as they did before.

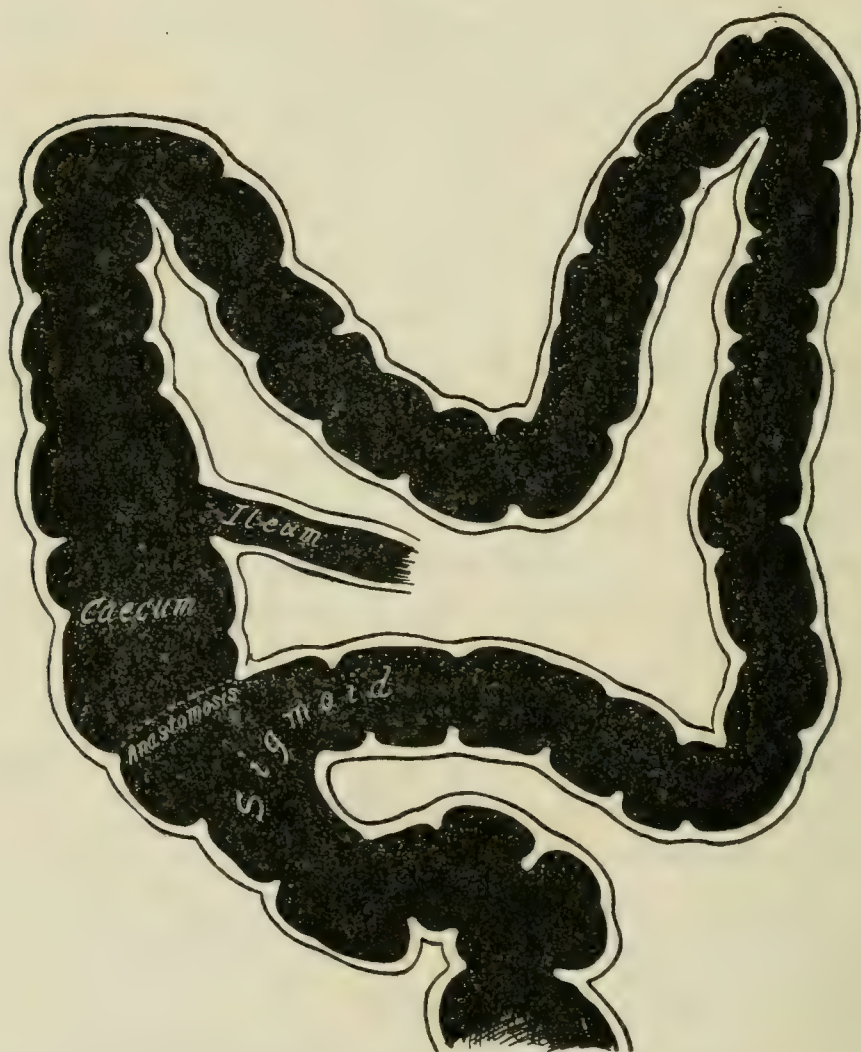
The principle is well worth remembering. I have had it illustrated in another instance in a case in which Doctor Sears and I were associated.

The patient came to me, also having had a cæcosigmoidostomy for constipation. X-ray showed that there was a perfect circle and no evidence of obstruction. The patient, however, was suffering from a decided intestinal toxemia. I turned her over to Doctor Sears for medical treatment. He treated her for several months but could never make much head-way. Finally he returned her to me for a surgical operation of some kind. X-ray pictures seemed to indicate that the fecal contents would come down through the ileocæcal valve, around through the ascending, transverse and descending colon into the sigmoid from the sigmoid back into the cæcum and form a vicious circle.

I opened the abdomen, not knowing just what operation would be done. I found that the ascending colon was very much dilated, also the anastomotic opening and neighboring sigmoid. I decided, then, to remove the ascending colon and the first half of the transverse colon.

With a purse-string we turned in the distal end of the transverse colon and turned in the cut end of the ascending colon, leaving practically no ascending colon above the ileocaecal valve. This patient was relieved of her caecal stasis by this operation and was made a perfectly normal woman so far as her bowels were concerned. (Figs. 27 and 28.)

FIG. 27.



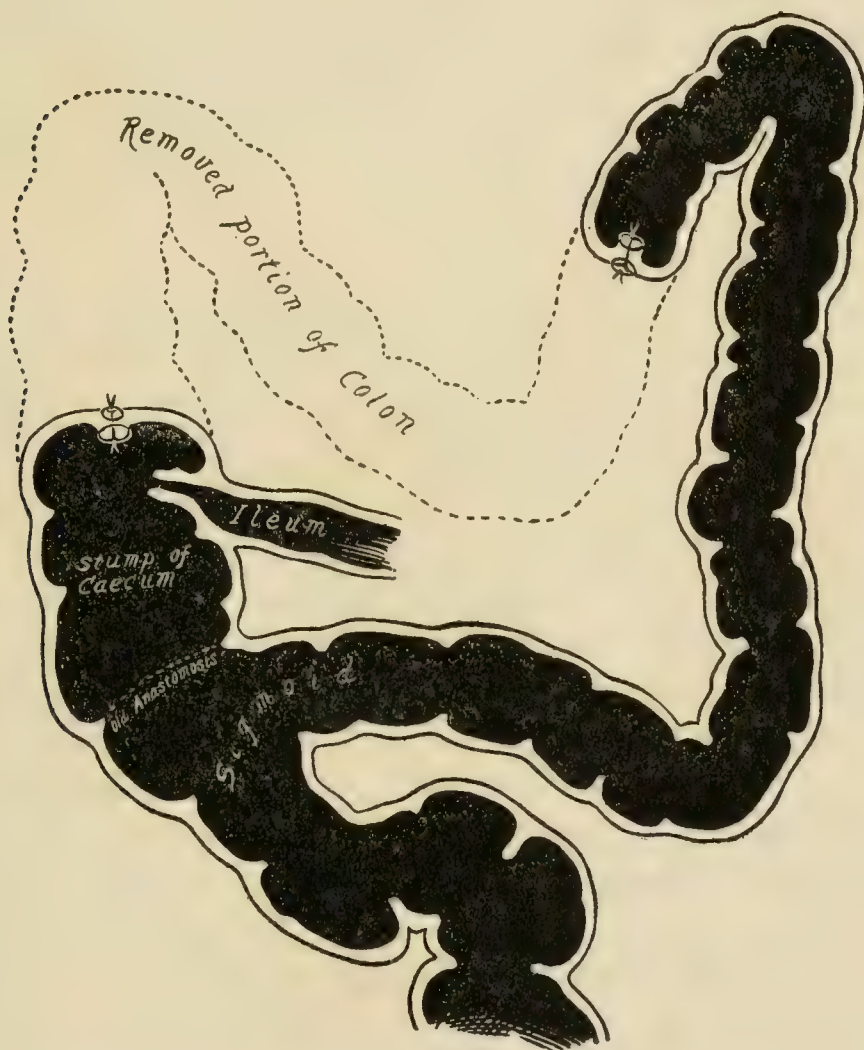
Vicious circle following cecosigmoidostomy for constipation.

This was the first time we ever combined a cecosigmoidostomy with removal of the ascending and part of the transverse colon. This operation was done more than six years ago and the patient has been entirely well since.

Some years ago it will be remembered that a number of men, particularly Eastman, of Indianapolis, recommended cecosigmoid-

ostomy for intestinal stasis or constipation. The results were not gratifying and the operation was not a success in most of the cases. I think if those men who performed a number of these operations would go back over their cases and remove the ascending colon and part of the transverse colon they would probably cure their patients in the same way that we cured this one.

FIG. 28.



Correction of vicious circle by removal of the ascending and transverse colon.

In doing intestinal surgery we must remember that the peristaltic wave normally runs toward the anus. Some have condemned lateral anastomosis, on the ground that the end of the bowel often dilated and made a very pathological condition. It is my opinion that only one end of the bowel dilates and that is the turned in proximal end, in which the peristaltic waves come down by the anastomosis

and continue on to the end of the bowel with the drive. It is plainly to be seen that this powerful intestinal contraction might finally dilate such an end. Therefore, in doing intestinal surgery, in which a lateral anastomosis has been done, the proximal end should be turned in by repeated sutures and made as short as possible. And when possible it should be covered in with omentum to further strengthen its wall. I still believe that lateral anastomosis is safer than end-to-end anastomosis. At least it has been safer in my work, both experimentally and clinically. Therefore, we should take notes of all the defects in lateral anastomosis and try to correct them.

**SURGICAL CLINIC—RIGHT OVARIAN CYST WITH
TWISTED PEDICLE COMPLICATED BY SALPIN-
GITIS AND INJURIES OF THE PELVIC FLOOR; IN-
JURIES OF THE PELVIC FLOOR RESULTING IN
CYSTOCELE AND RECTOCELE, WITH HEMOR-
RHOIDS; CARCINOMA WITH RECTOVAGINAL FIS-
TULA COMPLICATED WITH LARGE FIBROID TUMOR**

By CHANNING W. BARRETT, M.D.

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Illinois College of Medicine; Gynecologist and Head of Department of
Gynecology, Cook County Hospital, etc.

CASE I. *Case History.*—Austrian woman, 42 years of age. Married. Has borne children. She comes in with an operating-room diagnosis of tubo-ovarian abscess, and salpingitis. She complains principally of burning in the right lower quadrant, leucorrhœa, headache and backache.

Onset and Course.—Patient states that she does not feel sick, but that she comes in on account of the burning in the right lower quadrant, which has been present for four months, gradually increasing in severity. In the last two months, she has not been able to do her housework readily, because of the pain and burning in the side, but she has not been laid up in bed on this account.

The leucorrhœa has been present off and on for a long time, for years. She states that it has never been profuse enough to cause great inconvenience.

Headaches have been present in the last four months almost constantly. They are located in the frontal region. The backache has been present for four months, but has not been severe, or troublesome.

Menstruation began at the age of nineteen years, regular 28-day type. Duration from three to five days. Quantity moderate. No pain. Last period September 17-21.

She has had five pregnancies, the first at twenty-one years of age and the fifth at forty-one years of age. The first three pregnancies

resulted in full-term deliveries. Normal delivery, puerperium normal. Usually gets up in four or five days. Has had two miscarriages, the first at forty years of age, and the second at forty-one years of age. Both at three months. Was married at the age of fifteen.

Separated from her husband four months ago on account of non-support.

Family history negative. No loss of weight. No particular disturbance of the bladder or kidneys. Bowels regular, appetite good.

Upon examination, the patient is found to be a white woman forty years of age, weight about 140 pounds. Does not appear markedly ill.

Head: Eyes normal, pupils react to light and distance. Ears, nose and scalp negative. Mouth and teeth: Upper absent, lower few. Tonsils present. Neck: Slight enlargement of thyroid, which has been present for many years. Chest: negative. Heart: negative.

Abdomen, moderately full, tense and thick-walled; slight tenderness in the right lower quadrant, especially over the appendical region, and down to the pelvic brim.

Liver, spleen, and kidneys not palpable. No masses felt in the abdomen. Extremities negative. Reflexes normal.

Pelvic examination: External genitalia show gaping vulva, with appearance of rectocele. Entroitus admits three fingers readily. Levator muscles palpated, widely separated. Wall distensible and deep. Cervix points downward, enlarged, softened and slightly patulous. Freely movable. The body of the uterus slightly retroverted and slightly increased in size.

Adnexa: Right, rounded elastic mass, about the size of a small orange, between the uterus and the pelvic wall. Seems fairly well defined, spherical, and separate from the uterus, showing some mobility. Slightly tender. Left tube, and ovary apparently normal.

Diagnosis.—Right ovarian cyst, possibility of moderate salpingitis, complicating.

Remarks by Doctor Barrett during operation.—The salient point in connection with this patient is that she is a woman forty-two years of age. Four years ago she began to suffer from a burning pain in the right lower quadrant of the abdomen. At about the same time she began to have headache and backache, and has had

leucorrhœa, the leucorrhœa being of a longer date than four months. She has not been laid up in bed with this pain in the abdomen, but she has been kept from her work a good deal.

We find upon examination that she has a rectocele, and gaping of the vulva and vagina, the levators are markedly separated. We find the uterus slightly displaced backward and slightly enlarged, and at the right side of the uterus is a mass, rather well defined and rather distinct in outline and almost too movable to be a pelvic inflammatory mass even though it were confined to the tube and ovary.

We want the head low on the operating table. We want an incision long enough to do the work well, but not longer. The person who would just as soon have an incision seven inches long as three inches long is thinking of the patient's abdomen and not of his own. We want an incision just long enough to do good work. Every extra inch that you put upon the incision increases the patient's chances of a disability.

This mass on the right side could be either an ovarian cyst or a tube. We think its movability is too great to be a pus tube.

Now, as we cut down upon it, we find that movability. We find the cause of a good deal of the burning and distress that this is causing. The pain is one point in favor of the diagnosis of salpingitis, but we find here the cause for her distress. Here is the ovarian cyst, with a twisted pedicle, but not twisted enough to cut off circulation entirely. This has not been greatly enlarged as a result of the twisting. How do I know that that has not been enlarged? It does not show the œdema and discoloration that an ovarian cyst with a twisted pedicle does in case of obstruction, and also the tube which has a portion outside of the twist, but does not show the enlargement. An ovarian cyst with a twisted pedicle that cuts off circulation will soon turn a blackish gray, and become œdematous, and will show a degenerated wall, and marked enlargement, and the tube that is outside of the twist will become very much œdematous, and very much swollen, enlarged, and will look very much the size and appearance of a child's open hand, a baby's hand. The fimbriæ stand out to some extent. There are some adhesions here, and there is a little evidence of mild salpingitis.

We also find in the anterior wall a fibroid. We will have to decide whether we will take the uterus out on account of the fibroid

before we undertake to do anything with the ovarian cyst. We find on the other side adhesions to the tube, but a fairly healthy ovary. There are adhesions to the sigmoid and to the broad ligament. There are adhesions of the omentum to the anterior surface of the broad ligament.

This patient is forty-two years of age. She is past the child-bearing period. She has quite a considerable fibroid in the anterior wall, and a little one in the posterior wall. So we will decide to remove the uterus.

In order not to produce a sudden artificial menopause, we will leave the left ovary which is in fair condition, taking out the tube, which is somewhat inflamed.

After grasping the broad ligament between the tube and ovary with a reliable clamp and carrying the incision just under the tube, toward the uterus, we put another forceps on the broad ligament, and the utero-ovarian ligament, and cut down, and grasp the vessels on the uterine side as they are bleeding. We shall expose the vessels running alongside of the uterus and grasp those with the forceps, thus securing them, and then cut those.

On the right side, we take hold of the untwisted broad ligament, under the outside of the tube and ovary, and cut down to the uterus, stripping the peritoneum to uncover uterine blood-vessels. There we find the blood-vessels running down beside the uterus on this side. We clamp those.

We find the incision on the right side, not very far from that on the left side, only the breadth of the cervix intervening and we cut across the peritoneum. The main vessels are all clamped. We may have bleeding from some vessel within the uterus. We shall take the uterus off just above the cervix.

We take No. 2 or No. 3 catgut and tie the uterine artery that we clamped, running alongside of the uterus. In tying that, we throw the point of the forceps upward, instead of pulling out upon the handle. That exposes the whole stump that we wish to tie. We have the uterine artery, on the left side and next, we take the uterine artery on the right side, which you see exposed.

Turning out on the point of the forceps, instead of pulling up on the handle, we tie. On the right side we take the broad liga-

ment, the round ligament, with the ovarian artery, and in this bite we leave the catgut long enough for traction.

We then go to the other side, in which we have left the ovary, and we now tie the round ligament, and the utero-ovarian ligament. Inasmuch as that is on in a way so that it might slip, by not getting a good, big bite, we will go around that once or twice, so that there will be no slipping of the ligature, and we will tie it again.

We will tie the broad ligament that lies between the ovary and the tube. Now, we will sew the peritoneal edge over and over.

We have here the stump. We take a catgut doubled, and take a bite of the posterior flap of the uterus, which we have made, a bite of the angle on the left side, the stump of the broad ligament, and the round ligament, a little bite of the angle anteriorly, and then a good bite of the anterior flap. Now, we draw the ligaments right in between the anterior and the posterior flap of the uterus, and cut the ligature on the broad ligament.

We come to the right side, and with a double ligature, the same as before, taking a good bite of the posterior flap. The tension is rather marked on the upper part of the broad ligament. We regrab that and tie it with a separate ligature.

We tie the broad ligament, and here is the upper border of it. You will notice that because on pulling out the ligature, it has allowed the ovarian artery to bleed into the tissues a little. Instead of one ligament in between, we will have two, the upper part of the broad ligament and the round ligament, separately. And we sometimes tie them in that way. Sometimes, if the round ligament does not lie right close, we tie them separately.

We return to our work of putting in the suture which unites the anterior and posterior flap. We have taken a bite of the posterior flap. We have a bite of the angle. We take a bite of the broad ligament, which slipped out when we made tension on it before. We take the round ligament on which the tension is not so great. We take a bite of the angle in front in order to keep the peritoneum from sagging off, and then a good bite of the anterior flap. Now, we draw rather carefully these ligaments in between. We draw on the broad ligament a little more. The tension on it is rather marked.

(Upon further tension the ligature broke.)

Here we are having more than the usual trouble with this side.

We will start in again and put in the ligature which broke.

Just as though we were starting anew, we take a bite of the posterior flap, a bite of the angle, a bite of the broad ligament, of the round ligament, of the peritoneal angle, and then of the anterior flap. We will try to get the approximation a little better, so that we do not have to pull so hard upon our ligature to secure it, so that the tendency to break will not be so great. We do not have to pull down so hard on that, and we will expect it to hold.

We cut the ligatures on the broad ligament and the round ligament, and we have the anterior and the posterior lips closed leaving only a short distance down from one broad ligament to the other.

We take a bite of the posterior flap, then a bite of this pedicle, which comes in between the anterior and the posterior lip, and then a bite of the anterior lip; then of the posterior flap again, of the broad ligament stump, so as to keep it inside smoothly, and the anterior flap.

We are now beyond the stump, and shall unite the anterior flap with the posterior flap, for this one and one-fourth to one and one-half inch distance. Then we sew back over this to smooth up any irregularity. And then we tie the stump again.

We have the peritoneum entirely closed over the stump, the anterior and posterior flaps coming together and closing, and the broad ligaments from the sides coming in between the flaps, to give support to the cervix and the upper part of the vagina.

We have seen a good reason for the patient having pain in the right lower quadrant, but she has had trouble in the region of the appendix, and we want to know whether it is caused by the tube and ovary or the appendix itself, and we find the appendix a very much thickened, and clumped organ, and so we will take it out.

We clamp and cut the adherent mesentery, so that we get the appendix to stand up perfectly free from the bowel. We clamp it at the junction of the bowel, so that we get all of the appendix out. We tie it at the clamped junction. We tie that because once in a while we get bleeding from the stump, and once in a while, in the effort to turn that in, we get a little leakage of material. We start with a purse-string suture, about three-eighths of an inch from the tie, on the cæcal wall, and we start opposite the

mesentery. We go in and out around the stump, but we are in at the mesentery. That is, we have the mesentery structures which have been cut, outside of the suture, because what is outside of the ligature now, as we have gone around here, will be inside and tied, as we turn them in.

Now, we have ties ready; we make traction upward on the purse-string, as we cut off the appendix. We now have it ready to turn in. Having got the appendix out, it is held with a toothed forceps, so that the assistant can let loose upon that and allow it to turn in. We will just touch this, but where we touch it, we will not take the sponge off and touch it some other place.

Now, then, taking a bite just at one side of the suture, on the side opposite the mesentery, we tie and start sewing, and finally cut the purse-string. We will reinforce this by sewing over it again, and by taking very superficial bites of the bowel wall.

Some are inclined to tie the appendix off and drop it back into the abdomen, without any turning in. We do deny but that most of these patients will get well; otherwise operators would not do it. But I would not be inclined to deal with a gunshot wound of the intestines by picking up the edges and tying a ligature around the edge, to see if they would get well. And yet we must admit that if we cut the appendix off right down to the bowel, that we have very much the same sort of condition as though a bullet had penetrated the bowel without tearing it.

We now tie the mesentery stump to the flap we have cut off. It had adhesions which bound the appendix down into the pelvis, and we oversew the stump a little, so as not to get any slipping of the ligature, and tie this catgut that we oversew.

We are now through in the abdomen. We replace the structures as nearly as possible, putting the cæcum back into position by bringing the omentum down properly into the field of operation. There is no oozing from the field of operation. The little blood that has gathered in the posterior cul-de-sac it wiped out.

In bringing down the omentum, we are careful to straighten it out and not to produce overtraction upon it. It is a good deal more important that the transverse colon stay up than it is that the omentum come down.

We have now closed the peritoneum. The fascia above that

runs down farther. This we must bring together. Some operators have taken the position that the important thing when closing the abdomen was the aponeurosis. The aponeurosis down at that area consists of all three of the fat muscles—the external oblique, the internal oblique, and the transversalis; none of this aponeurosis going posterior to the recti muscles. They have insisted that the important thing was to close the aponeurosis, and that no importance was to be attached to the closing of the recti muscles; in fact, that it was harmful to undertake to sew them.

But I would not take that position now. We do not wish a hernia to exist through the abdominal wall, as far as the aponeurosis. It is true that the aponeurosis might stop the hernia, but it is also true that it might not, and we do not want the hernia to extend even that far. We do not want the recti muscles continually grinding upon the abdominal viscera. In fact, if we should have a diastasis recti, we would count it rather important for the patient's welfare to open the abdomen, even with a long incision, to unite the recti muscles.

And so, one step in the procedure of closing the abdomen, is to bring together the recti muscles.

Now, then, there comes the question of what further we shall do with this patient? She is now beyond the probable child-bearing age, even though the uterus were not removed so any degree of weakness of the pelvic floor may be corrected.

At forty-two years of age, she has reached the period of life that we could decently estimate that she is half through with her life. That is, we could fairly estimate that she might have forty-two more years to live. She is a good hardy woman. It would not be strange if she lived to be eighty-four years old. But supposing it is thirty years instead of forty-two. She is entitled to those thirty or forty or forty-five or fifty, whatever she has in store, to be as good years as possible. If we leave the pelvic floor the way it is, she will carry a fair percentage of disability all the rest of her years, if it does not amount to an absolute disability. She will lack the strength to work or to exercise or to do the things that she wants to do, being on her feet. She will grow old by reason of the weakness that is brought about in this way. She will grow old from disinclination to take proper exercise, there will be a greater tendency to sit, a greater tendency to get out of the proper position, and not to carry

the proper poise, an inclination to let the abdominal walls sag down, so as to take off any strain upon the pelvic floor.

We believe that a large percentage of women should have the disability of the pelvic floor repaired, when they have reached the end of the child-bearing period, if they have not been forced to have it done before that time. And so, added to this procedure will be the repair of the pelvic floor, to leave her in good condition to carry out the activities that she may see fit to carry out.

Now, it is true that a woman forty-two years of age, or at the age of middle life, may not take advantage of exercise, and may not take advantage of straightening up, may not lay the proper weight upon the importance of doing so, even though she is put in condition to do that. But she is in a position, if the pelvic floor is repaired, to carry on those activities, if she can be made to see the importance of them. And a part of our work should be to try to make her see that. A part of the success of surgical work done upon a patient depends upon following them up with the proper after advice.

The steps to be taken in this procedure are based on the proposition that the pelvic floor is part of the abdominal wall. It is the lower wall. It is, therefore, the most important part of this wall, and that by reason of its being the lower part of the abdominal wall, it needs to be the strongest part of that wall, or it would be, if it were not for the openings through it for physiologic purposes.

The woman is at a great disadvantage, in having to have the physiologic openings through that part of the abdominal wall, which should be the strongest. When she has an injury to her pelvic floor, she has an opportunity for a hernia and the structures coming down through that opening constitute a hernia.

Now, just immediately upon recognizing this as a hernia, it occurs to us that no skin plastic operation, no mucous membrane plastic operation is going to do the work, that if this is really a hernia through the pelvic floor, then we must deal with the essential anatomical structures of the pelvic floor to cure it. That is, if we have a hernia through the anterior abdominal wall, we have to deal with the muscles composing the walls of the abdomen. If we have a hernia here, which we have, we want to deal with the muscles which constitute the the pelvic floor, and the muscles here of importance are the levator ani muscles.

At a certain place is the hymen just posterior to the duct of the Bartholinian gland. The same on the other side. Pick up a portion of the labia minora at the hymen just posterior to the duct of Bartholin. Then we catch this in the centre posteriorly. We raise the tissue lying between the forceps on the one side and the median incision into a ridge, and with a knife we follow it down the line of junction of the labia minora, the vestibule and the hymen. It is important that we get that at just the right place. This line draws in unusually. So we will catch the scar tissue and hold it up. Otherwise, we will cut off tissue which the patient has a right to keep. This incision is what the initial incision is to laparotomy. It is simply for the purpose of gaining access to the part.

We now catch this flap in the center, and this on each side (indicating) and we have control of the flap. We will put a little sponge into the vagina to keep back any secretion. Holding the flap taut, we cut right along the edge.

If we are not particular to keep close to the edge, we will encounter the rectum. So we keep close to the edge, of the vaginal flap, being careful not to cut through it. It would not be a serious thing if we did, because that part is going to be taken off anyway, but it might interfere with the traction upon the flap.

As we have cut back a little ways, we push that down with the sponge, take a better hold on the flap, than we could in the beginning.

We now push that back, and any fibres of tissue which hold, we cut, and then with a sponge we push it back; and we finally come to the smooth area, this pocket-like area, which shows now. Then two fingers are introduced into that, and we push that out, and we uncover the levator ani muscles.

With a good solid, good-sized needle, threaded with No. 2 or No. 3 catgut, holding the rectum back, and putting the levator muscle on the stretch, we sweep around the levator ani muscle with the needle, taking a large bite of that muscle.

We are rather particular not to draw out a little portion of the levator ani, as some have described, getting a little part of the muscle, but leaving the rest, but we go around the whole border.

After taking a bite in the flap, introducing the finger under the levator ani, we put the needle point at the end of the finger, start it in the tissue on the left side, and then withdraw the finger and push

back the soft parts above the levator ani muscle. Now, we cross, holding the rectum back again out of the way, so as not to get a bite of the rectum, and go around the border of the levator ani muscle on the right side; then across to the left side again, and taking a bite of the levator ani muscle farther back. We then sponge this out, and raise the flap, and then with the forceps in the median line, pressing backward upon the rectum, we tease the levators together, not with any difficulty, or strain, but perfectly easily, and tie them.

We now have the levators coming together, between the vagina and the rectum. The levators are felt, not lying at the side of the vagina, but coming together between the vagina and the rectum.

But the same test on that shows that it is not coming forward far enough to cure the hernia. Lifting the flap, we push the levator ani muscle away for a distance, and on the opposite side the levators may now be felt running off to the bone on the two sides. They must be picked up and they must be united farther forward. We take the fascia above the muscle, the muscle, and then the fascia beyond the muscle. Then we take a bite of the flap, in order to fix it so that it will not have any tendency to slide, the fascia above the muscle, then the muscle, and then the fascia below the muscle. That is, it would be below the muscle if the patient were standing on her feet. The fascia which we spoke of as above the muscle is the recto-vesical fascia, and below and anteriorly are the two layers of the triangular ligaments.

We bring those together. We test this again, and we find that it is closed enough now to give the patient good support. We take off the excess of vaginal edge, and then with silkworm gut, on a cervix needle, we start at the end of the hymen on our right side, where we took hold, and gather little bites of the flap, threading it on the needle just as you would thread an angle worm on a fish hook, being careful not to get the bites too large or too irregular, or getting out on the mucous membrane surface, and coming out here and taking the end of the hymen at that side, one forceps on the two ends, and holding it right up in the center.

The deeper structures are sutured, starting in right close to the superficial ones, going in down to the muscle, taking a small bite of the muscle. We come back on the opposite side and extending out so that

the suturing on the two sides will be uniform as to the amount of tissue engaged, and as to the location where it comes out, and about the same distance from the raw surfaces on the two sides.

In tying, we take first, the purse-string suture that took the two ends of the hymen, and tie it. That tie restores the hymen, or what is left of it. We cut off these little tags that come down between. Next comes the suture, which takes in the deeper structures. We tie from before backward, tying the purse-string suture first, and then the most anterior deep ones, and so on back, keeping them uniform. And by reason of having made the incision at the right place, not too far out; we have the vestibule here (indicating); we have the posterior vestibule in the normal condition, and the hymen restored as much as there is any of it left, and inside we have the levator muscles coming across, to make a good supporting structure.

This patient, by reason of this sag, has had an enlargement of the hemorrhoidal veins and it will be a disability if we allow that to go on. There is rather a transverse shaped group of veins, and so we cut through transversely, and unite the skin to the rectal wall. We will insert a small piece of gauze.

The uniting of the levator ani muscles takes away the lifeless appearance of the vulva and pulls that (indicating) in there; it gives it a lively look again and a normal appearance.

CASE II.—This patient is a woman fifty-seven years of age. She is a widow. She was referred to me from the outside for hemorrhoids. The interesting thing about this was that it was rather difficult to get this patient admitted to my service from the outside, because she suffered from hemorrhoids. A hemorrhoidal case goes to surgery. But after quite a good deal of effort, they had her transferred to my ward. This is the condition:

She complains of painful defecation, bloody stools, small, pencil-like stools, and frequent urination.

Onset and Course.—Patient states that she has been having hemorrhoids off and on for the last twenty-five or thirty years. She has had an acute attack of hemorrhoids, of about one week's duration. She complains of painful defecation. The pain is intense when she has a bowel movement, so severe that she compares it to child-birth. Between bowel movements, she has intense burning pain in the rectum.

The bloody stools have been present for the last week. She states

that a small painful tumor projects out of the rectum after defecation. This protrusion she has to replace. She complains of frequent urination, which comes on in attacks at varying intervals of time. She has to urinate about every half hour, in small amounts.

Sexual History.—Menstruation began at twelve years of age. Regular twenty-eight-day type. Duration, five to eight days. Quantity, very profuse. No pain. Had the climacteric at thirty-six years of age. Had three pregnancies, one at seventeen, the last at twenty-two years of age. Full term. Spontaneous delivery. Patient thinks possibly she was lacerated at second delivery. Normal puerperium.

This patient complains almost entirely of the hemorrhoids, and yet she gives a very definite history of disturbance of the bladder. Upon asking her to strain down, things begin to roll out, and there is a rectocele and a cystocele; and when she reaches a certain point in this rolling out, the urine begins to flow.

There are some portions of the bladder sphincter present, because there is no complaint of inability to hold urine. The support of the bladder is gone. The patient begins to strain and the bladder begins to roll out, and we get more and more of a cystocele. When this reaches a certain point, the force she uses overcomes the sphincter control, and the sphincter action ceases to exist, and the urine comes away.

When we get the structures which belong above the levator ani, coming down below, we have a hernia. That is, we have an organ which should be in the abdomen, getting outside of the abdomen. The structures of the pelvis which belong above the levator ani muscles, to start from below, are nearly all of the rectum, all of the bladder, some of the urethra, and uterus, most of the vagina, which, however, should be mentioned before the uterus, and the peritoneal cul-de-sac, and the intestines. Those should all be above the levator ani muscle.

In these cases it is not at all uncommon to find below the levator, all of the urethra, a good part of the bladder, quite a good part of the rectum, practically all of the vagina, and a portion of the uterus, sometimes all of the uterus, and sometimes the peritoneal cul-de-sac and sometimes the intestines.

This patient has quite a marked hernia. She has quite a large cystocele and rectocele coming down, a good deal of hernia of

the bladder, rectum and vagina, and yet it is doubtful, from an examination without an anæsthetic, if the uterus comes down very much. You might say that that is an advantage. It would be an advantage if it were free; but she complains of a good deal of rectal trouble, and it seems that this uterus is rather adherent to the rectum.

What should we do with this, if it does not come down? To make her surgical treatment complete, it would call for perhaps an abdominal hysterectomy, and then an anterior colporrhaphy, a rather extensive operation upon the anterior vaginal wall, to hold the bladder up, and then a perineorrhaphy, and then a hemorrhoid operation. That is too much of an operation for a woman in this condition. She is fifty-seven years of age, and looks older than her age, and should not have so much operating at one time, if we can help it.

If the uterus came down well, so that it was almost an outside operation vaginally, we would be inclined to do a vaginal hysterectomy, and then an anterior colporrhaphy and perineorrhaphy, and rectal operation, may be all at one sitting.

When you have to do two procedures, that of going into the abdomen above, and all this work below, it is rather extensive work in the condition in which this woman is. So we will be inclined to correct the vaginal conditions at this time. And then if she has trouble, we will do a laparotomy at another sitting. Two safe operations are always better than one very dangerous operation.

We may open the posterior cul-de-sac and free this small uterus, which is no doubt very atrophic. She had her menopause at the age of thirty-six years, and now she is fifty-seven. We may undertake to free this uterus from the rectum, so that it ceases to press upon, if possible, and we may avoid a second operation in that way.

I am saying that with the idea that the uterus does not come down well. When we get her under the anæsthetic, it may be a different proposition. We may then find that the uterus comes down well enough to do a vaginal hysterectomy, but I think not.

It is easily demonstrated here, what I meant by my last observation on the other patient, that is, there is a lifeless condition and appearance here. No muscle pulling in there, holding it or giving it any

appearance of life. There is a little bit of a uterus there. It is hardly as adherent as I had figured. There is an ulcer. There is a hemorrhoid of considerable size, but there is an ulcer here, and she must have those attended to. The small uterus is well up, and a little adherent. I do not think it is going to be as troublesome as I had apprehended. So we will do nothing with that. We will simply attend to this cystocele, and the pelvic floor and the hemorrhoids.

We first take hold of the cystocele in the middle, at the projecting part, then make just a little incision, being careful not to cut into the bladder. By just making that little incision carefully, we can then introduce the knife handle anteriorly through that and push the bladder off. Then, with that pushed off, there is not the danger of getting into the bladder that there would be in dissecting.

Then we enlarge that up toward the uterus. Wherever the bladder holds to the vaginal wall, we make a little dissection with the knife, and keep on pushing the bladder away.

Now, with a sponge on the finger, to increase the friction, we push this off. Now, we have this down close to the cervix, and we enlarge the incision up toward the urethra, until we are close to it. Then we notice there is a condition of the urethra which must be attended to also. Here we have the urethra (indicating). Then comes the bladder. We mobilize the bladder from the vagina, so that we can take off as much of the vaginal wall as we wish to. We take that off transfixing the flap and running the knife posteriorly and anteriorly, taking out the portion of the vaginal wall that we do not want. We cut through here (indicating) and enlarge posteriorly, and then enlarge anteriorly, without any risk of getting into the bladder, because the separation has been made.

Then we carry that back into position, leaving a long elliptical incision to be closed. We will simply hold this down to the extent necessary to put in the first suture, and as soon as we have got it sutured enough, so that any of it can be pushed back, we let it go back where it belongs. We are doing this work to have the vagina held up. And so we do not wish to hold the uterus and the vagina in the position that we are trying to correct while we are doing it. We like to have it just as readily as possible take the position that we wish it to maintain.

We now get to the vaginal wall and the connective tissue and the muscular fibre and the structure that still exists, in these bites, and get a bite on the other side. There is a peculiar combination in this fascia, in that it has no strength and no elasticity. That is, she has not the proper support, and yet the tissues have so lost their elasticity that while they roll down and give her a great deal of trouble, yet there is no room to work. There is no give to the pelvic floor.

We are closing this, getting as good and solid a structure as we can. We will have this so that it can not roll down over the pelvic floor. We must depend upon this procedure to hold the bladder up.

Here is a lot of thickened tissue. I will go right into the urethra, taking off that scar tissue. If I cut that off as high as I want to, and let it go, it will retract too much, so that we will put the suture in before cutting it off. Now, we have the urethra fastened, and we will get rid of a lot of hard connective tissue in that region.

We have now dealt with the cystocele, and the urethrocele, and a rather extensive urethral caruncle. We have now got things in such condition that if we construct a complete pelvic floor, they will not come down.

We have a lot of scar tissue to deal with here, which must be removed. The vestibule must be left. We now take just the edge, and then this edge (indicating). If we get much deeper than the edge, it will crowd us in our dissection, too close to the rectum. We must be very careful about every adhesion of the old scar tissue, where the vaginal wall was torn.

Now, having got it started, we roll it out in this way. We now begin to get a smooth area. This shoves the rectal wall altogether out of the way. Now, we have a smooth area, and with that smooth area we can easily push this off aside, knowing that the rectum is back, and that we are not interfering with it. We can cut in high enough to get at the levator muscles.

With good strong catgut, and with the flap held forward, and the rectum crowded backward, that muscle is put on the stretch. We let this strong needle encircle that muscle, and we make it come out before letting go of it. We now take a good bite of the flap. The needle is put in above the levator muscle, with the rectum held back, and then with the needle straight, we shove so that we press

away from the muscle, and come out around the muscle. Then we hold the rectum out of the way, and come back and get a good bite of the muscle again. Here (indicating) is soft tissue. We get a good bite of the rectum on that side. We sponge out. The flap is held forward. The muscles are made parallel, and we hold the rectum out of the way and tease those together.

Upon feeling, we find that instead of the old condition, we have a good muscle, coming from bone on each side, up to the median line. But we wish to unite this farther forward in order to actually cure the condition of the patient, so that it does not roll down, and so that she gets support. So we push the muscle off the flap. Then, lifting the flap up, we take a good bite of the muscle on this side. It was not that this patient was lacking in muscle, but the muscle was located in a position where it was not doing the patient any good.

There (indicating) is the fascia thickened, and then there is the muscle in between, and there is the adhesion above, the recto-vesical fascia and the triangular ligament. These muscles come together with just a little tension, and in order to bring them together well, I have to make tension. Now, this extra amount of flap is taken off.

We place the suture from before backward, in their order.

The question has often arisen as to whether—and it has often been decided in the negative—as to whether a hemorrhoid operation and a perineal operation should be done at the same time. I say that we do not hesitate to do a hemorrhoid operation after doing a perineal operation.

We take this mass off in a way that will take in the ulceration, if possible. If we want to take off a little tumor mass at some other point, we cut it off and sew the edges together. We would not cauterize it. That used to be the method in amputating fingers, and it has continued a good deal longer as the method of amputating piles.

A portion of the pile bearing area is to be excised. It is done by clamping, cutting, and tying. With that under control, we take off this second big, thickened mass of pile tumor.

We pack the rectum with iodoform gauze.

CASE III.—We have one case that I want to show you to-day, a case on which we are doing a third operation, instead of merely a second one. She had a carcinoma between the rectal and vaginal wall, an extensive carcinoma with recto-vaginal fistula. Un-

fortunately, we have had to contend with a large fibroid tumor, so that we could not do an extensive operation through the vaginal route, obliterating the vagina and rectum, as would be necessary, to get rid of the carcinoma lying between the rectal and vaginal wall, so that the first procedure that we did upon her was to remove the large fibroid abdominally.

After she recovered from that, the next procedure was to take out the sigmoid and a portion of the rectum, down low on the rectum, and to unite the lower end of the descending colon to the abdominal wall, so that she had an artificial anus on the left side, doing that work through a medium incision, and then bringing the gut out through a puncture incision, you might say, on the left side.

We inverted the cut end of the rectum down quite a distance, so that when we come to do the vaginal work, the rectum will be reached below. We took out about eighteen inches of bowel and united the lower end of the colon to the abdominal wall.

The next operation we will have to do—she recovered well from that operation except a temporary paralysis of the left foot—will be to dissect out the rectum and the vagina obliterating these outlets and stop hemorrhage, which will not be any trifling matter here, with the carcinoma going on.

That is not at all ideal, but it is not common to have a carcinoma in this region. We have made the artificial anus beforehand, for one reason, that she will have all the operation that she can well stand probably in doing this obliterating operation from below, and, next, we have wanted the artificial anus beforehand, so as to eliminate the secondary conditions that are present with the carcinoma, that is, the inflammatory conditions that are there by reason of the fecal matter always going over this carcinomatous area.

I have some doubts about getting a radical cure of the carcinoma after doing all three of these operations, but it was laid before her and she elected to do her part toward getting such a result as we were able. The radical operation has since been performed and the patient has returned to hard work and suffers very moderate inconvenience with the artificial anus.

A SURGICAL CLINIC

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HYPERTROPHIC VILLOUS SYNOVITIS OF KNEE-JOINT— SYNOVIAL CAPSULECTOMY

F. B., male, aged 26, carpenter, was admitted to Medico-Chirurgical Hospital on January 15, 1921. The family and venereal histories are negative.

The patient states that five years previous to admission the right knee-joint became very painful and much swollen. This swelling subsided, but reappeared two years later, when it was aspirated at another hospital. Three months previous to admission he fell down, injuring the right knee, which again began to swell. He was again treated at another hospital as an ambulatory case: A plaster-of-paris splint was applied to the knee and allowed to remain for nine weeks, and when it was removed the joint once more began to swell.

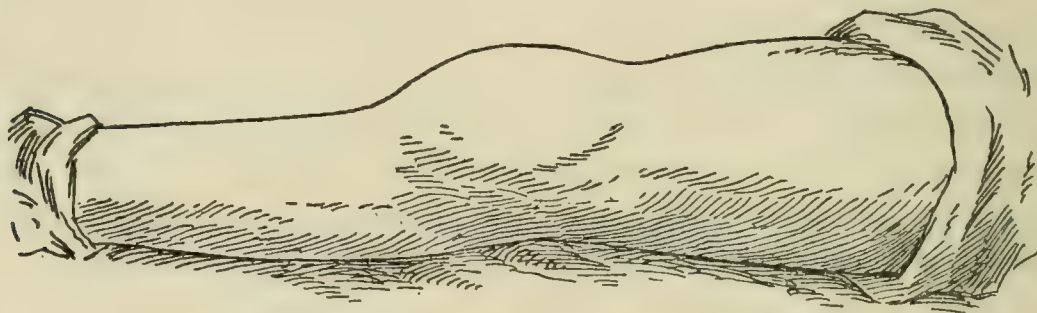
Physical examination of head and trunk negative. Right knee shows exudate in synovial pouch (Fig. 1) with increased local heat over same. X-ray report by Dr. G. E. Pfahler: "The examination shows evidence of a well-marked osteoarthritis indicated by exostoses growing from the edges of the joint, exudate into the joint and an exudate into the bursa under the quadriceps tendon. There is also some general infiltration of the tissues about the joint, such as occurs in arthritis. There is no evidence of fracture showing at the present time."

Here is a case of hydrops articuli which was first aspirated and then the joint immobilized for a prolonged period, neither of which measures prevented recurrence. There was nothing in the history suggestive of injury to the semilunar cartilages, crucial ligaments, tibial spine or convex articular surfaces of the femur, nor was there anything in the history or findings suggestive of constitutional disease (tuberculosis, syphilis, metastatic infection from a primary focus). The patient had obviously reached a point where radical measures

were indicated. His temperature at the time of admission was 99 3-5°F.

Operation (January 18, 1921).—A horseshoe-shaped flap of skin and fat, and with convexity below, was outlined (Fig. 2) and dissected upward. The quadriceps tendon, patella and ligamentum patellæ were divided by the Z-shaped method advocated by the late J. B. Murphy (Fig. 3) and retracted, revealing the hypertrophied and deeply congested villi as portrayed by the colored illustration (see frontispiece). The picture well shows how the elongated villi were caught between the joint surfaces during movements of the knee, pinched and irritated, giving rise to the clear straw fluid that escaped

FIG. 1.



Appearance of swollen knee-joint previous to operation.

when the joint was opened. Synovial capsulectomy was performed, the villi-bearing synovial membrane being separated and removed from the subsynovial layer: During this dissection bleeding was free, but was controlled by hot compresses and forcipressure. Further exploration revealed no underlying injury or disease of any of the cartilaginous, bony or ligamentous structures of the joint. The osseo-aponeurotic anterior wall of the joint was now closed by interrupted sutures of No. 2 chromic gut, the patella fragments being approximated by suture of the quadriceps tendon tissue streaming down in front of them. The skin and fat were apposed by interrupted sutures of silkworm-gut. A rubber-dam drain was placed in each angle of the superficial incision, so as to drain the subcutaneous space. A dry, sterile gauze dressing and bandage were applied.

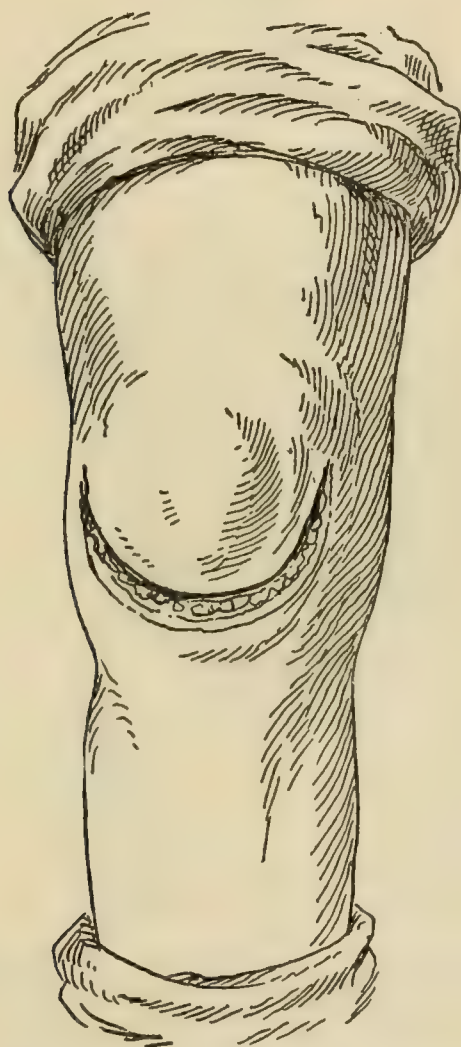
Postoperative Notes.—January 19—the day following operation. Temperature, 100; pulse, 72; respiration, 16. Suprapatellar bursa

aspirated, 50 cc. of sero-sanguineous fluid being withdrawn. Buck's traction apparatus applied to right leg. Drains removed.

January 21st—Aspiration repeated.

January 27th—(9th day).—Sutures removed: Healing *per primam*.

FIG. 2.



Transversely curved "horseshoe" incision through skin and fat. The lowermost point of convexity falls midway between patella and tubercle of tibia. When the flap is raised the whole thickness of the fat is taken with it to insure a good blood supply.

February 1st—Buck's traction apparatus removed. Patient encouraged in active motion of knee.

February 18th—Patient walks about with aid of crutches.

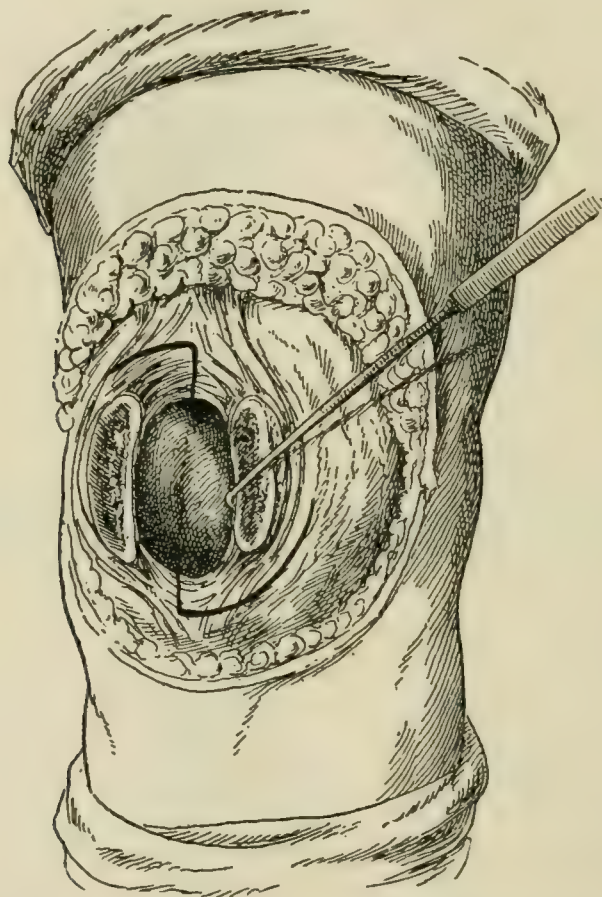
March 18th—Crutches discarded; cane substituted.

March 26th—No pain; no swelling. Girth of right knee, 38 cm.: of left, 37. Patient can flex knee to right angle.

April 4th—Discharged from hospital, cured. Motion good and patient walks without stick.

June 15th—The patient has gone back to work. Since the operation there has been no swelling of the joint, nor has there been any pain in the joint. Complete extension is attainable, while the range of flexion is about 100 degrees.

FIG. 3.



The Z-shaped division of the quadriceps tendon, patella and ligamentous patellæ advocated and employed by Murphy. Much freer exposure of the joint for exploratory purposes is obtained by this than by the midline incision of Jones. When restoration is made, the more the quadriceps muscle contracts the more firmly the cut patella surfaces are approximated.

Hypertrophic villous synovitis of the knee-joint has not received much attention in the literature—at any rate, not under that caption. The late Dr. John B. Murphy reported 2 cases (*Murphy's Clinics*, vol. v, No. 1, February, 1916, pp. 155-170). The etiology is variously given as recurrent trauma (acting from without or from within the joint), low-grade non-tuberculous subsynovial infection, and subsynovial tuberculosis. It is obscure. The synovial membrane undergoes hyperplasia and becomes congested, so that hydrops occurs.

Villous formation takes place, those villi in relation with the tibial articular surface become pinched when the knee is in action, and some become detached, producing rice-bodies. The constant friction may in time bring about erosion of the articular cartilage of the femur, with eventual destruction of same. The semilunar cartilages may become thickened and raised up by underlying pockets of exudate, and may also undergo attrition from the movements of the joint; in fact, the joint may be completely wrecked. After ablation the synovial villi can be beautifully demonstrated by floating the specimen out upon water.

The chief sign is recurrent hydrops of the joint. The patient suffers little or no pain, but is incapacitated in his work and in locomotion. After aspiration palpation may give the impression of a doughy mass within the joint. Chills, fever and other signs of frank infection are absent; in fact, physical examination suggests a mechanical rather than infectious cause for the hydrops, very much as in the hydrops due to injury of a semilunar cartilage.

The diagnosis here, as in so many obscure affections of the knee-joint, is most satisfactorily established by exploratory arthrotomy.

As to the treatment, Murphy states that no measure will effect a permanent cure short of excision of the hypertrophied synovial tissue (synovial capsulectomy) with the possible result of a stiff knee temporarily, later performing arthroplasty, if ankylosis result, to give the patient a movable knee; after having performed capsulectomy one cannot accurately prognosticate to what extent the range of motion will eventually be restored. Murphy, and also Volkmann, have referred to the frequency with which these joint-changes occur in patients with a hemorrhagic tendency; my case, as the history shows, exhibited a decided tendency to bleed, and profusely. In order to lessen the oozing the foot of the bed should be elevated 18 inches.

FRACTURE THROUGH ARTICULAR CARTILAGE OF FEMUR AT KNEE:
ARTHROTOMY

D. M., male, aged 18, machinist, was admitted to hospital on June 19, 1921. The patient states that on January 24, 1921—five months previous to admission—while at work he jumped down 3 feet on to a plank, which gave way, and he landed on his left knee, which was thrown into flexion. He got up and found his knee out of control,

and a lump appeared on the *outer* side. The joint soon began to swell. He was put to bed for 10 days, but no traction was applied. At the time of admission he is able to walk on the knee, but it plays out; it slips and throws him and remains swollen. Something slips at the side and the patient goes forward; if it goes back with a distinct click the patient is able to bend his knee again; otherwise he cannot.

Physical examination of the left knee shows no apparent swelling; the landmarks and bony prominences are clear and distinct. There is a tenderness (a "sickening" sensation) on pressure over the anterior third of the lateral surface of the external condyle; less over the internal condyle; still less over the ligamentum patellæ. X-ray examination negative.

From the history and findings injury to the external semilunar cartilage was suspected, but as the internal semilunar is usually the one that is involved, and as the case was not wholly clear, exploratory arthrotomy was advised.

Operation (June 21, 1921).—Same exposure of joint as in previous case. Exploration revealed on the outer face of the external condyle—just below the outer patellar ridge—a fragment of cartilage-covered bone, the size and shape of a small lima bean, broken off and remaining movable within the limits of a few fibrous bands that held it against the femur. The synovial membrane in relation with this fragment as a center was red and velvety from irritation; there was, however, no excess of synovia in the joint. The cartilage-covered bony fragment was removed. Both semilunar cartilages were carefully examined, but found intact. The wound was closed with a subcutaneous rubber-dam drain in each angle. Buck's traction was applied.

Postoperative Notes (June 22).—Suprapatellar bursa aspirated, yielding about 20 cc. of sero-sanguineous fluid. This fluid was examined bacteriologically, but proved sterile.

June 28th—Stitches removed; healing *per primam*.

July 1st—Buck's traction apparatus removed.

July 4th—Patient sent home to-day, two weeks after operation. He has been encouraged in employing active movements of the knee, and states that for the first time since the accident he is able to move the joint without pain.

July 14th—Patient is getting about on crutches. The range of

motion in the joint is steadily increasing. Massage and baking are instituted. A cane is substituted for the crutches, thus preparing the joint for passive motion, which will be begun after the lapse of two weeks. It is better to have several weeks of active motion before passive motion is begun.

Roux-Berger (*Am. J. Surg.*, 1921, xxv, 54) describes a case in which there was a fracture through the articular cartilage, and the fragments acted as free foreign bodies: In this case wide exposure by a U-shaped incision was necessary to clear up the diagnosis.

These two cases—both examples of uncommon conditions—emphasize the importance of free exposure of the joint, and therefore of exploratory arthrotomy. If one should start out with a preconceived diagnosis of injury to a semilunar cartilage, make the usual limited and confined incision for the removal of the latter, and not find the cartilage definitely injured, one would have to close and then make the free exploratory arthrotomy incision. Again, as pointed out by Smith, a true lesion of the semilunar cartilage may be associated with a tear in the anterior crucial ligament. It is therefore conceivable that ablation of the semilunar cartilage alone without further exploration has been responsible for a certain number of failures even when a torn semilunar was definitely repaired (Roux-Berger). Midline splitting of the quadriceps tendon, patella and ligamentum patellæ may be considered a rather formidable procedure: In fact, it does look formidable at the operating table, but the wound heals promptly and without any permanent injury to the joint. Better than the midline incision of Robert Jones, however, is the Z-shaped method employed by Murphy (see Fig. 3), which affords more satisfactory exposure of the deeper recesses of the joint.

Objection has been raised to the transversely-curved superficial flap on the ground that gangrene might occur when the flap is dissected to some distance above the patella. Gangrene is not apt to occur, however, if one has the lowest point of convexity of the flap opposite the mid-point between the patella and the tubercle of the tibia, and also if one takes care when raising the flap to dissect the fat cleanly from the underlying deep fascia and aponeurosis, thus safeguarding the superficial blood-vessels.

Immobilization in plaster-of-paris is not indicated after exploratory arthrotomy of the knee, provided the anterior crucial ligaments

are intact, for it militates against the early resumption of motion by the joint. Buck's traction is indicated for about ten days to keep the synovial surfaces apart, to counteract muscle contraction, and to effect a compromise between absolute immobilization and unchecked mobility.

If hemarthros appears after operation early aspiration should be done. (Consult article by Fisher in *British Journ. Surg.*, April, 1921, p. 500.)

LIPOMA OF NECK: ABLATION

On June 13, 1921, a man 35 years of age presented himself with a swelling in front of the neck. This swelling was most pronounced in the left submandibular region, whence it diffused itself downward and over to the right of the midline. The overlying skin was normal in appearance, although somewhat stretched by the swelling. On palpation the swelling was soft, even semifluctuating: The growth was diffuse, and its borders were poorly defined. There was nothing to suggest lymph-node origin. The swelling presented some of the characters of a cold abscess, but the patient was robust and had had the condition for several years with no disturbance other than that of the contour of his neck. Cervical extension of a ranula was simulated, but there was no primary ranula in the sublingual space. Branchial cyst was considered, but this swelling was too superficial and gave the impression of lobulation. The diagnosis was therefore made of a soft, diffuse lipoma, which variety of neoplasm when it occurs on the front or sides of the neck is characterized by its soft, semi-fluid consistency.

Operation (June 14, 1921).—Although the most salient portion of the tumor lay high up under the left angle of the jaw, the incision was not placed directly over it, for it was thought that by making a collar incision and elevating the superficial flap well upward this large lobule could be dislocated and shelled out without difficulty. Accordingly, the transverse crease just below the pomum Adami was selected as the site for the incision and was infiltrated intradermally with one per cent. procaine solution. Deeper subcutaneous infiltrations were made over the area occupied by the tumor. The flap, consisting of skin, superficial fascia and platysma, was dissected upward almost to the symphysis menti, exposing the lipoma, which lay upon the deep

fascia, but not beneath it. The tumor was removed from below upward, the prominent globular lobule in the left submandibular region being readily reached and shelled out with the handle of the scalpel. Bleeding was trifling in amount. To obliterate the dead space between the platysma and the deep fascia the anterior edges of the platysmæ were approximated by interrupted sutures of No. 1 plain gut. The skin incision was closed by interrupted sutures of silk-worm-gut, and a rubber-dam drain was placed in each angle of the wound. A dry, sterile gauze dressing was applied and retained by a bandage. At the close of the operation the skin was somewhat reduntant, loose and baggy.

Postoperative Notes (June 15th).—Rubber-dam drains removed.

June 21st.—Sutures removed. Healing *per primam*. The loose and baggy skin had retracted, so that the front of the neck was now smoothed out. The scar was very inconspicuous and was hidden by the collar. Thus, a scar that would have been noticeable was not substituted for the tumor, which was of practically no pathologic importance—at least not enough to warrant leaving a conspicuous scar.

As to the similarity between a lipoma in this region and a cervical extension of a ranula I need but refer to a paper I wrote about two years ago, entitled "A Contribution to the Surgical Pathology of Ranula," (*Surg. Cynece. & Obstet.*, vol. xxix, No. 5, November, 1919, 447). "Physical examination revealed in the left submandibular region a swelling that was not tense, but soft and fluctuating, which presented many of the characteristics of a cold abscess, namely, by the absence of the cardinal signs of inflammation, aside from the swelling. The skin over the swelling was normal in color, freely movable, and free from adhesions and indurations." Note the difference, however: "On opening the mouth there was seen beneath the left side of the tongue—in the alveololingual sulcus—a cystic swelling that bulged more and more as the submandibular swelling was pressed upon. On releasing the submandibular swelling from pressure the sublingual swelling subsided to its former size, thus suggesting a direct channel of communication between the sublingual and submandibular swellings."

Branchial cysts affect more commonly the right side of the neck and occupy a deeper plane than a lipoma. The swelling presented by

such a cyst is described in a case of Gilman's "Branchial Cysts and Fistulas," (*Journ. Amer. Med. Assoc.*, vol. 77, No. 1., July 2, 1921, 28). "The neck showed a prominence below the angle of the jaw on the right side, over which the skin appeared normal. The swelling was about ten cm. in diameter, was prominent and fluctuated, giving on palpation an impression of a cyst with thin walls containing thick, semifluid material under moderate tension. The outlines of the swelling were indefinite, and it was evidently covered not only by skin and subcutaneous tissue, but also by several deeper layers."

Medicine

THE MANAGEMENT OF DYSPNŒA DUE TO DISEASE OF THE CIRCULATORY SYSTEM*

By ROBERT BABCOCK

Chicago.

THIS is in reality cardiac dyspnœa, for it is a matter of common observation that persons with extreme arteriosclerosis, chronic nephritis or valvular disease may experience no uncomfortable shortness of breath so long as the myocardium remains functionally competent. Dyspnœa is only a symptom but since its treatment is often unsatisfactory it may assist us in our therapeutic measures to get as clear an understanding as possible of the mode of its production and therefore a brief account of the most rational theories to explain it may not be out of place.

Among the early theories advanced to explain cardiac dyspnœa was that of acidosis. Excepting in cases of diabetes or nephritis together with myocardial incompetence it is coming to be accepted that some other condition than acidosis must be sought to account for the symptom here discussed since it does not yield to alkalization of the blood. That an excess of the carbonic acid in the blood supplied to the respiratory center is the cause of the hyperpnœa experienced by cardiopaths goes without saying, but the real problem calling for solution lies in the observation that notwithstanding the increased rapidity of breathing the sense of dyspnœa does not abate. Some explanation of this fact must be sought therefore.

In an article on cardiac dyspnœa which appeared in Nelson's *System of Medicine* of January, 1921, Peabody advocates the mechanical theory by which he means diminished pulmonary ventilation and lessened vital capacity of the lungs. It is essentially the view to which R. G. Pearce arrived as a result of his studies carried out in Cleveland and described in his article in the *Archives of Internal*

*Read at a meeting of the American Clinical and Climatological Asso., June, 1921, at Lennox, Mass.

Medicine of February 15, 1921. By experiments on himself and others Pearce demonstrated that during periods of excessive physical efforts there was, notwithstanding greatly increased respiratory rate and acceleration of the pulse rate to 160 per minute, a decreased alveolar ventilation so that the volume output of the heart was not able to furnish sufficient oxygen to the respiratory center and tissue cells in general. In other words the greatly augmented metabolism was out of proportion to the exchange of gases in the lungs and to the ability of the heart to receive and deliver the necessary supply of oxygen. It is conceivable accordingly that when in consequence of cardiac disease the blood-carrying power of the heart falls below the oxygen demands of the body cells the excess of carbon dioxide in the blood stimulates the respiratory center to abnormal action and dyspnœa results.

In this connection it is interesting to note the view expressed by Hoffman in Nothnagel's "System of Medicine," vol. 14, regarding the influence of pulmonary congestion in the production of the emphysema observed in some cases of chronic heart disease. He based his views on the experiments of Grossmann in von Basch's laboratory. This investigator found that when pulmonary stasis was created in dogs it was followed by a lowered position of the diaphragm, an increase in the volume of the lungs and a diminished pulmonary expansion in consequence of which condition alveolar ventilation was lessened notwithstanding the acceleration of respiratory movements. There resulted as Hoffman considered a rigidity of the lungs and mechanical interference with normal exchange of gases in the alveoli. Hoffman accordingly preceded Peabody and Pearce in the advocacy of the mechanical theory to account for cardiac dyspnœa.

If now we apply the facts brought out by the experiments of Grossmann and Pearce to our cardiac patients suffering from shortness of breath we have the following sequence of events; despite the increase of heart rate decreased volume discharge of blood per minute in consequence usually of some defect valvular or myocardial of the left heart; impediment to the blood-flow through the lungs with a corresponding tendency to stasis in the pulmonary vessels which stasis eventually extends to the veins of the greater circulation with consequent loss of equilibrium between the arterial and venous systems;

defective gaseous exchange within the tissues and inevitable increase of the carbon dioxide in the blood carried to the lungs and respiratory center. Breathing now becomes accelerated but fails to produce adequate alveolar ventilation in consequence of the shallowness of respiration caused by the tendency to rigidity of the lungs and by defective play of the diaphragm.

Respiration becomes accelerated and assumes more or less strikingly the costal type. There is inspiratory retraction of the epigastrium with more or less evident narrowing of the costal angle described by Hoover in pulmonary emphysema and some cases of chronic heart disease. It is plainly apparent that despite increased respiratory effort the lungs do not expand sufficiently to meet the patient's desire for air.

Physical exertion enhances the dyspnœa and hence the first indication is to confine the individual to bed. Tissue metabolism is thus decreased, less carbon dioxide is generated, venous flow to the heart is less rapid and the over-burdened organ is given a chance to slow down, thereby receiving and discharging a larger volume of blood per minute. Gaseous exchange within the alveoli is facilitated and correspondingly more oxygen and less carbonic acid are carried to the respiratory center with proportionate lessening of dyspnœa. The next indication as I apprehend it is to attempt so far as possible the restoration of circulatory equilibrium; that is, lessen venous congestion and improve arterial flow. Two means are at our command in addition to absolute physical and mental repose, namely, digitalis and hydrogogue cathartics. Some therapeutists resort at once to digitalization of the patient so as to get the heart under control as soon as possible. Then when its full physiological effect is obtained the remedy is withdrawn. Doubtless this procedure accomplishes the result in most cases, but experience has proven its danger in cases of myocardial degeneration. Consequently I believe in achieving the desired effect more slowly and safely and that is by attacking the venous stasis while at the same time endeavoring to slow down and energize cardiac action.

In the early years of my practice I read assiduously the works of well-known English and Scotch authors. They were great clinicians because compelled to rely on close study of their cases and of the action

of remedies. They did not possess and hence did not depend on our modern instruments of precision and on that account were better doctors. At all events we owe very much to their teaching as regards both diagnosis and therapeutics of cardiac disease.

From Balfour, Bramwell and especially Fothergill I learned that no therapeutic measure more surely surpasses profuse watery catharsis for the relief of cardiac dyspnoea when associated with hepatic congestion as is usually the case. This has been proven over and over again in patients with mitral stenosis. The query is often made by doctors as well as members of the family "can the patient stand it? Will it not cause too great weakness?" As a matter of fact the circulatory disorder and rapid breathing produce more exhaustion than purgation can possibly do as demonstrated by the comfort and easier respiration following the first half dozen watery stools. This beneficial action I have always explained on the theory of the mechanical improvement of blood-flow resulting from lessened engorgement of the hepatic and intraabdominal veins. Very likely that is an important factor, but some experiments recently reported by Professor Luckhardt of the physiological laboratory of the Chicago University suggest another explanation as also possible.

Professor Luckhardt's experiments consisted in isolating the lungs of frogs and then stimulating the distal end of the pneumo-gastric nerve. The effect of the vagal stimulation was an intense blanching of the lung supplied by the nerve stimulated. He found further that, if the exposed lung was atropinized by being painted with a strong solution of atropia, stimulation of the pneumo-gastric failed to blanch the lung.

The Professor has thus demonstrated the existence of vasomotor nerves in the coats of the pulmonary vessels and has settled a controversy that has raged for a century. Now the possible bearing of these experiments on cardiac dyspnoea is this. The swollen liver of hepatic stasis is not only sensitive to palpation but in many instances is painful so that the sufferer sometimes attributes his dyspnoea to the distress in his stomach. May it not be possible that painful hepatic congestion acts as a stimulus to the vagus with consequent constriction of the pulmonary vessels? If such be the case, alveolar ventilation would be correspondingly diminished. Granted such an

effect, then profuse catharsis should lessen or abolish vagus stimulation and by just so much tend to relieve cardiac dyspnœa.

According to Huchard there is still another reason for the administration of cathartics to persons suffering from cardiac dilatation and consequent stasis within the capillary and venous systems. In his opinion digitalis proves more effective after free watery discharges have emptied the heart's chambers and blood-vessels of some of their extra load. In accordance with this view I have preceded or associated the use of digitalis by purgation and believe that in this way I have more surely and speedily obtained the desired result, namely, diminution of venous engorgement and a slower more efficient cardiac action. When possible the digitalis is given intravenously until the heart is brought under control. Moreover the dosage of digitalis is determined not by the body-weight but by close observation of its effect as shown by lessened dilatation, increased vigor of systoles and decreased congestion of lungs, liver and systemic veins. Further detail in the use of the remedies mentioned would be to weary the patience if not insult the intelligence of this audience.

In the cases in which dyspnœa is greatly intensified by extensive œdema with hydrothorax and ascites the foregoing measures may not prove efficacious for speedy relief and more energetic ones must be employed as paracentesis and a diuretic. Tapping is generally without risk if carefully done, but acute pulmonary œdema has been known to follow the too rapid withdrawal of hydrothorax. Its main drawback lies in the fact that the relief it affords is usually transient unless followed up by theocin or diuretin or a drastic purgative like elaterin. If the kidneys are not too badly damaged they will generally respond to diuretin in sufficient dosage, not 40 to 60 grain *per diem* as I have known many physicians to prescribe but 90 to 120 grains in twenty-four hours and continued for several days. My original experience with this remedy was in 1891 in the case of a man who could not be made by any means known to me to pass more than sixteen ounces of urine in twenty-four hours. On diuretin in doses of fifteen grains every three hours day and night he soon passed eight quarts a day with entire disappearance of his dropsy and dyspnœa. His was a case of arteriosclerosis of the great internal vessels with myocardial break down. Diuretin is disagreeable to take but in my experience

does not upset the stomach so quickly as theocin. I always order a fresh preparation from an unopened bottle and give it in solution in cinnamon tea or water to which essence of pepsin is added by the nurse. The original manufacturer, Knoll, stated that when exposed to the air diuretin lost much of its solubility and administered in capsules was more likely to disturb the stomach.

When all these measures fail we have no other resource than to relieve the dyspnœa by morphine or other opium derivatives subcutaneously. Indeed we often have to prescribe an opiate while endeavoring to restore cardiac efficiency and remove venous stasis as already indicated. Permit me only to add that Luckhardt's experiments suggest wisdom of combining atropine with the morphine, and I think I have gotten better results from this combination than from morphine alone.

Lastly it may be stated that a Carell diet of milk not to exceed eight ounces four times daily greatly assists the treatment just outlined. Of course the length of time during which this rigorous mode of treatment is to be maintained must be determined by results, but in most instances offering any hope of restoring compensation dyspnœa can be overcome in a few days. Then in some persons comes the real difficulty of management by which is meant the further restriction or control of the individual who begins to chafe against restraint since his distress has been relieved. The importance of such restriction for many weeks or even months is shown by the liability of some cases, especially of mitral stenosis, to recurrence of dyspnœa upon resumption of the previous occupation or habits as to exercise which many persons with limited cardiac reserve erroneously think necessary for the maintenance of good health. As a matter of fact persons with weak hearts feel better and stronger when they do not exceed their physical, that is, cardiac limitation. Compensation having once broken may be restored by judicious management in many instances, but with each succeeding break the restoration of functional efficiency becomes more difficult and less certain. Cardiac patients should be taught therefore that "an ounce of prevention is worth a pound of cure." Mitral stenosis of extreme degree, that is, the so-called button-hole mitral is particularly incompatible with much physical effort. The integrity of the right ventricle and of the left auricle is indis-

pensable to cardiac efficiency and when these chambers dilate dyspnœa is sure to supervene. Such cases should be kept under frequent observation and the individual put to rest as soon as signs of failing circulation appear. In these and indeed in all other cases displaying symptoms of failing heart power the persistent use of digitalis should not be neglected. It is well also to order a periodic purge for the purpose of combating the ever-present tendency to pulmonary and general visceral stasis. For such persons "Eternal vigilance is the price of safety."

**A CASE OF DIABETES MELLITUS IN WHICH THERE WAS
EXTREME PANCREATIC FIBROSIS AS WELL AS A
TUMOR OF THE ROOF OF THE FOURTH VEN-
TRICLE OF THE BRAIN**

BY F. PARKES WEBER, M.A., M.D., F.R.C.P.

WITH A PATHOLOGICAL REPORT BY HUBERT M. TURNBULL, M.D.,
Director of the Pathological Institute of the London Hospital.

THE patient, H. S., an English watchman, aged 52 years, was admitted to the German Hospital under Dr. Parkes Weber on the evening of November 30, 1920. He was in a comatose state on admission and remained more or less deeply comatose till his death on the following evening, December 1st, at 10 P.M. The history, as obtained from the patient's wife, was that he had complained of lassitude and depression for some weeks; he had a great appetite and had been extremely thirsty. He continued at his work till a week before admission, when he had to give up because of increasing feebleness. On November 28, 1920, at 3 A.M., he was seized with a convulsive (epileptiform) attack, and on that day a doctor saw him. After the attack speech was at first regained, but from the morning of November 30th he became gradually more lethargic, till he was admitted to hospital during the evening of that day in a condition of coma.

The patient's temperature in the hospital was 96° - 97° F.; the pulse was 84-88 per minute, till the afternoon of December 1st, when it was 96; the respiration was 16-20 per minute, till the afternoon of December 1st, when it was 34. Brachial systolic blood-pressure (December 1st): 100 mm. hg. The urine (December 1st) was of specific gravity 1020; weakly acid; containing a trace of albumin and 3 per cent. sugar; Legal's reaction for acetone was positive; Gerhardt's reaction for diacetic acid was negative. Nothing special was noted by ordinary examination of the thorax and abdomen. The pupils were somewhat contracted and no conjunctival or corneal reflex was obtained (December 1st). The patient could take practically no food or drink by the mouth, and could not speak, though sometimes he

seemed to understand what was said. A sodium bicarbonate solution was prescribed to be given per rectum twice daily, and nutrient enemata were ordered.

On December 1st his first epileptiform attack in the hospital occurred. The clonic portion of the convulsion was followed by such a relatively long death-like cessation of respiration, that the nurse came downstairs to say that the patient had died. However, natural breathing recommenced, and his condition appeared the same again as it was before the convulsion. I then gave him an intravenous injection of about 120 c.c. of a sterilized alkaline saline solution, but for some reason I could not inject more. About mid-day another epileptiform convulsion occurred, this time in my presence, and the clonic phase of the fit was followed by a pause in respiration (over a minute ?), so that death really seemed to have taken place. We pressed the chest two or three times and pulled up the glottis, and spontaneous breathing gradually recommenced. Between this time and 4.30 P.M. five more convulsive seizures occurred; they were similar to the preceding ones, but the period of death-like cessation of respiration at their termination was less striking. After 4.30 P.M. there was no further fits, and death occurred at 10 P.M. Artificial respiration was tried in vain.

POST-MORTEM EXAMINATION ¹

The heart weighed 9 1-2 ounces and showed nothing special. There was some hypostatic congestion of the lungs. The liver was rather large, and on section the ordinary fine markings were not very distinct to the naked eye. The kidneys were somewhat small and ordinary macroscopic examination suggested the presence of slight chronic interstitial nephritis. The pancreas was shriveled and weighed only about one ounce. Nothing else special was observed at the necropsy, excepting that there was an irregularly shaped lobulated *tumor of the roof of the fourth ventricle of the brain*. I took the mid-brain and the atrophic pancreas to Dr. H. M. Turnbull (London Hospital), whom I have to thank for the following careful pathological report.

I think it clear that the patient suffered from diabetes mellitus connected with extreme atrophic fibrosis of the pancreas, and he

¹ For assistance in this I have to thank the house-physician, Dr. Fankhauser.

probably died from real diabetic coma. Yet the remarkable terminal series of epileptiform convulsions—each convulsion followed by a period of death-like cessation of respiration—may have been in some way induced by the (gliosarcomatous) tumor of the roof of the fourth ventricle. These periods of cessation of respiration following the clonic convulsive stage of the fits recall (as kindly pointed out to me by Dr. Henry Head) certain observations by Kussmaul and Tenner on epileptiform fits in animals, experimentally produced by bleeding.²

PATHOLOGICAL REPORT FROM DR. HUBERT M. TURNBULL

Two specimens were received in formaldehyde solution from Dr. Parkes Weber on December 3, 1920 (P. M. 650. 1919 Appendix).

I. The first specimen comprised the *mid-brain, pons and medulla*. The cerebellum had been removed by an incision through the right and left peduncles.

Macroscopic Examination.—The anterior and posterior extremities of the specimen are bent upward. A mass of abnormal tissue (Fig. 1.) conceals the floor of the fourth ventricle, with the exception of the anterior 0.3 cm. on both sides and a further lateral strip (1 cm. long and 0.4 cm. at broadest) on the right side; in the posterior extremity of this lateral strip, that is 1.3 cm. from the anterior extremity of the ventricle, the deepest depression of the right anterior fovea lies exposed. The mass which thus conceals the greater part of the floor of the fourth ventricle measures 1.9 cm. from before back, 1.5 cm. at its broadest, and 1 cm. in depth. Its dorsal surface shows some six lobular projections, each measuring about 0.6 cm. in diameter, and each of these is nodulated, the nodules measuring about 0.2 cm. in diameter. The mass can be turned upward and backward so as to expose the whole of the floor of the fourth ventricle, except a minute portion of the posterior extremity. The inferior surface is less lobulated and less nodular. The mass is attached to the lateral margins of the posterior part of the fourth ventricle as far forward as the *striæ acusticæ*, but it is not attached to the apex of the *calamus scriptorius*; a probe can, therefore, be passed forward from the *calamus*

² A. Kussmaul and A. Tenner, "On the Nature and Origin of Epileptiform Convulsions Caused by Profuse Bleeding and also of Those of True Epilepsy," English translation by E. Bronner, New Sydenham Society, London, Selected Monographs, 1859, vol. v., pp. 13-19.

beneath the mass and over the floor of the ventricle. In color the surface of the mass resembles the adjacent brain substance.

The mass resists the knife on section, and feels gritty. The cut surface is, like the outer surface, divided into lobules. Each of these lobules is subdivided into round or oval areas of fairly sharp definition, the oval areas being largest and measuring 0.2 by 0.1 cm. The areas are of a glistening, pearly white color, which contrasts sharply with the yellower, duller cut surface of the subjacent medulla. Those of round shape have a central, opaque yellowish dot, and those of oval shape a central, opaque yellowish line.

When the mass is turned back, the fourth ventricle is found to measure 2.2 cm. in length and 1.9 cm. at its broadest. The median groove in the floor is curved considerably, with its convexity to the left. A transverse section has been made through the floor, 0.7 cm. behind the anterior extremity. The depressions and elevation seen upon the normal floor are all present. The apices of the posterior foveæ lie 0.6 cm. from the posterior extremity. Immediately in front of these course the *striae acusticae*. The posterior limbs of the anterior foveæ commence 1.1 cm. from the posterior extremity of the ventricle.

The remainder of the specimen is normal in appearance, except that the ventro-mesial border of the right olivary body lies 1 cm. to the right of the mid-line, whilst that of the left lies 0.8 cm. to the left of the mid-line. The roots of the third cranial nerve on the right side and of the eighth on the left side are absent; the remaining roots are of ordinary size and show a white cut surface.

Examination with the naked eye reveals, therefore, no abnormalities except: The presence of a lobulated, gritty tumor which is attached to the margins of the posterior part of the floor of the fourth ventricle, excepting the apex of the *calamus scriptorius*, and which lies over the whole of the floor of the fourth ventricle except the small areas detailed above; a dorso-ventral bending of the whole specimen with concavity dorsalwards; a lateral bend with concavity to the right; slight lateral displacement of the right olivary body. These bends and the displacement of the right olivary body are probably the result of the fixation of the specimen in a bottle so small as to require breaking in order to release the contents. They can scarcely have been caused by the pressure of the tumor, because such pressure as was exerted by

the tumor has not obliterated the ordinary sulci and elevations on the floor of the fourth ventricle.

Microscopic Examination.—A vertical segment was removed from the bulb at the level of the posterior fovea, so as to include the tumor and its lateral attachments. Paraffin sections were stained in Ehrlich's hæmatoxylin with eosin, Weigert's iron-hæmatoxylin with van Gieson's mixture, Weigert's fuchselin with neutral red, and 3 per cent. silver nitrate with neutral red.

The section passes through the nucleus and intramedullary fibres of the hypoglossal nerve, the dorsal motor nucleus of the vagus, and the sensory nucleus of the vagal and glosso-pharyngeal nerves. The fourth ventricle is roofed over by tumor tissue, which is attached laterally to the medulla in the site of the teniæ (Fig. 2).

The tumor is composed of glial fibrils and nuclei. The glial fibrils are arranged so as to form a dense feltwork in the large rounded areas and a loosely meshed reticulum in narrow strands between the rounded areas. The tumor consequently has a distinctly lobular structure. The proportion of nuclei to fibrils varies greatly. In almost all the nodules the proportion of nuclei to fibrils is in parts decidedly less than in normal glia, but in other parts the nuclei are in great excess. In only a few of the areas in which the nuclei are most numerous can cell-bodies be distinguished. The tumor is, in the main, cellulo-fibrillar rather than cellular.

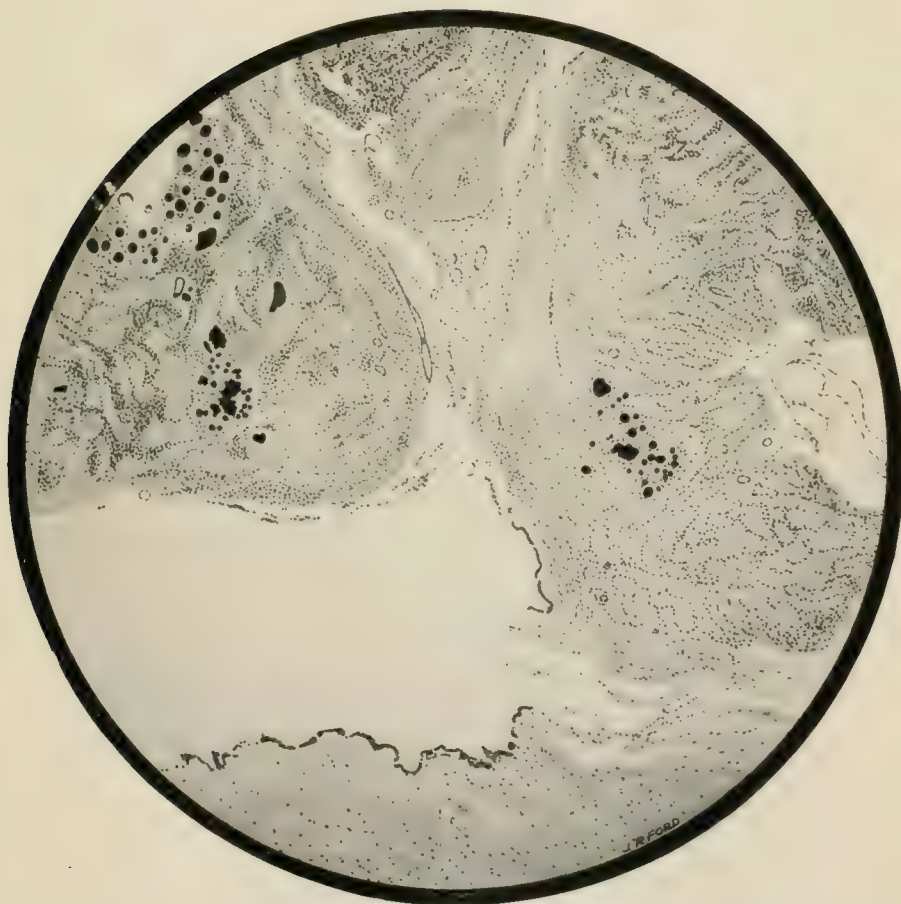
The vessels in the tumor are almost all cut transversely. The walls of the majority are greatly swollen, and are stained pink by van Gieson's stain. This hyaline degeneration is associated with narrowing or complete obliteration of the lumen. In several of the vessels, both occluded and not occluded, the hyaline wall is impregnated with granules of calcium. There is also a great number of calcareous bodies (Fig. 2). Most of these appear to be spherical, and in many, a paler central area is visible; one or more concentric lamellæ are sometimes present round the pale central area. A few are cylindrical and resemble longitudinally cut vessels. There are also large calcareous plaques, of which some are obviously formed by confluence of groups of the small bodies. The bodies tend to occupy the centers of the glial lobules. There can be little doubt that most, if not all, of these calcareous, psammoma, bodies are formed by calcareous impregnation of vessels which have undergone hyaline degeneration.

FIG. 1.



Specimen I, showing tumor lying over the fourth ventricle.

FIG. 2.



Gliosarcoma, with psammoma bodies, in roof of fourth ventricle. Hæmatoxylin and eosin. X30. The drawing includes the floor, lateral wall and roof of the ventricle on the right side. The inferior boundary of the tumor lies immediately above the transverse zone of reticular tissue at the lower end of the lateral wall of the ventricle. On the extreme right of the drawing, near the centre, is seen a portion of the outer surface of the tumor, covered by lepto-meninges.

FIG. 3.



Head of pancreas. Weigert's iron-haematoxylin and van Gieson's mixture. X18.

FIG. 4.



Tail of pancreas. Weigert's iron-haematoxylin and van Gieson's mixture. X28.

Where the tumor forms the lateral walls and roof of the fourth ventricle its inner margin is smooth and sharply defined. Where it forms the lateral walls it is lined by an almost continuous layer of ependymal cells; where it forms the roof a few small groups of ependymal cells are present at wide intervals (Fig. 2). Where the tumor meets the medulla the transition from tumor to normal tissue is remarkably abrupt. On each side the inferior periphery of a dense lobule of the tumor lies almost exactly in the plane of the floor of the ventricle, and a very narrow zone of loose-meshed glia lies between this and the subjacent nerve fibres (Fig. 2).

The bodies of the neurons in the hypoglossal nucleus are of sharp angular shape; they contain neuro-chromatic bodies which are sharply defined, and are evenly distributed, except in a few cases where a small portion of the cytoplasm is occupied by pigment; the nuclei are central. In the dorsal motor nucleus of the vagus the bodies of the neurons are seldom so angular; round the nuclei of the cells the neuro-chromatin frequently either is powdered, or is absent; fine pigment is present in a few cells. In the sensory vago-glosso-pharyngeal nucleus a few large cells are present and these are deeply pigmented. A few of the small cells are slightly pigmented. These small sensory cells have a central nucleus. When granules of neuro-chromatin are present, they are small, scanty and usually confined to the periphery of the cell. There are many "amyloid bodies" in this nucleus; they are very numerous and conspicuous above the nucleus in a zone beneath the floor of the ventricle. There is also in this sensory nucleus a slight proliferation of the glial cells.

Conclusions.—The tumor is a gliosarcoma of the roof of the posterior part of the fourth ventricle. It is cellulo-fibrillar, and shows little histological evidence of rapid growth. It contains numerous "psammom bodies," many if not all of which are the result of calcareous impregnation of hyaline, degenerated vessels. In the subjacent medulla there is no evidence of degeneration in the hypoglossal nucleus except in slight pigmentation of a few cells. The pigmentation in the cells of this and the other cranial nuclei is adequately explained by the age (52 years) of the patient; it cannot be accepted as a degeneration caused by the presence of the tumor. In the dorsal motor nucleus of the vagus evidence of slight degeneration is given by perinuclear tigrolysis and considerable loss of angular shape. In

the sensory vago-glosso-pharyngeal nucleus there is evidence of greater disturbance. Lack or sparsity of neuro-chromatic bodies in such small cells is common where special care has not been taken in fixation. Little importance can, therefore, be attached to this phenomenon here. But the presence of amyloid bodies and a slight proliferation of glial cells gives definite evidence of pathological change.

II. The second specimen consists of *pancreas and peripancreatic tissue*.

Macroscopic Examination.—The specimen has the general appearance of a pancreas rolled into a mass. A small portion of duodenum lies upon the head. When extended, the specimen measures 11 cm. in length, 2.4 cm. in breadth across the head, 3.2 cm. in breadth across the body and tail, and 1.5 cm. in thickness (from before back). It weighs, after fixation, 1 ounce.

Transverse sections show that the specimen includes the splenic artery and vein, a few small lymphatic glands, and a considerable amount of extra-pancreatic lipomatous tissue. The actual glandular pancreatic tissue revealed by section measures only: In head, 1.7 cm. from above down and 0.8 cm. from before back; in the neck, 2 cm. by 0.6 cm.; in the center of the body 2 cm. by 1 cm.; in the tail 2.6 cm. by 0.9 cm. It is obvious, therefore, that the weight of the glandular tissue is considerably less than one ounce.

The cut surface of the head shows sharply defined, rounded, white nodules, averaging about 0.2 cm. in diameter and separated by very delicate gray lines; lipomatous tissue isolates one group of nodules. In the neck slightly smaller white nodules are separated by more conspicuous gray lines. In the center of the body the nodules are again about 0.2 cm. in diameter, and are separated by conspicuous gray lines. In the tail a few ill-defined, pale yellow areas of the same diameter lie in a pearly white ground.

Microscopic Examination.—Complete transverse segments were taken from (1) the head, (2) the neck, (3) the center of the body and (4) the tail of the pancreas. Paraffin sections were stained in Ehrlich's hæmatoxylin with eosin, Weigert's iron-hæmatoxylin with van Gieson's mixture, Weigert's fuchselin with lithium carmine, Ehrlich's hæmatoxylin with mucicarmine, in dilute carbol-fuchsin,

by Gram's method with neutral red, and by Pearl's prussian blue method with neutral red.

(1) *Head of Pancreas*.—The interlobular septa are composed of densely packed, stout, collagenous fibres. There is also an interacinar fibrosis within the majority of the lobules. The degree of interlobular and interacinar fibrosis varies; the greater the degree, the greater is the loss of parenchyma (Fig. 3). The glandular tissue is, consequently, represented by one or two rounded groups composed of two or three large lobules, each of which is separated by a band of dense fibrous tissue, and by more completely isolated lobules. The isolated lobules show every degree of interacinar fibrosis and acinar destruction; the least affected are seen as rounded lobules within which the interstitial increase is confined to the border of the larger vessels and ductules, whilst the most affected have been converted into very small, rounded areas of dense fibrous tissue in which are embedded ductules and a few acini, or ductules alone.

The large ducts contain a few reticular masses and roundcretionary masses of mucus, together with a few desquamated epithelial cells and red corpuscles. The interlobular ductules contain threads of mucus.

Islands of typical structure and groups of insular cells are very numerous. They are present in all lobules, even the most fibrotic. They are sometimes surrounded by dense fibrous tissue, but there is no fibrosis nor degeneration within them. The centroacinar cells are remarkably large and conspicuous, being frequently cubical and occasionally columnar. The appearances suggest that groups of insular cells are formed by disappearance of the gland-cells and persistence of large centroacinar cells.

The interstitial tissue, in general, contains few fibroblastic nuclei and few infiltrating cells. The infiltrating cells are lymphocytes and plasma cells. There is little hypertrophy of the intima of the large arteries. There is no endophlebitis nor endarteritis. The interlobular septa contain numerous elastic fibrils of a tenuity which indicates recent development. Stout elastic fibres are present in their normal positions, but show some swelling and fragmentation.

(2) *Neck of Pancreas*.—The changes are similar, but the destruction of parenchyma is greater. The transverse section of the gland at this level is, consequently, remarkably small.

(3) *Center of Body of Pancreas.*—The changes are almost identical with those in the head. The bands of the interlobular fibrosis are slightly broader so that the remnants of lobules are more widely separated.

(4) *The Tail of the Pancreas.*—The fibrosis here is extreme. The cross-section of the gland is represented by an oval area of dense fibrous tissue; in this are embedded large ducts, and small oval and round areas which are traversed by a greater or less number of fibrils stained red by van Gieson's mixture. These small areas contain a few scattered ductules and acini and are densely infiltrated with small round cells (Fig. 4). Islands of typical structure are present in a few of these fibrosed, infiltrated remnants of lobules.

Some of the ducts contain large, mucous concretion.

The collagenous fibres in the greatly enlarged interlobular septa are slightly stouter and denser than in the previous sections; numerous delicate elastic fibrils are present among them. There is endophlebitis of a few venules, and endarteritis of a very few arterioles.

Among the cells infiltrating the remnants of lobules lymphocytes are in great preponderance; plasma cells and cells containing granules of iron are numerous; there are a very few neutrophil leucocytes.

Bacteriological examination was confined to two sections from this segment, stained with dilute carbol-fuchsin according to Pfeiffer's method, and two sections stained by Gram's method and counterstained with neutral red. A few Gram-negative bacilli were found; one measured 4.3 by 0.6 micra; the remainder measured from 2 to 2.6 micra in length by 0.5 to 0.7 micra in breadth. Two diplococci were also found in the sections stained in carbol-fuchsin.

Conclusions.—Throughout the pancreas a great fibrosis is associated with very extensive destruction of parenchyma. The fibrosis is the result of an inflammation of considerable duration, as is shown by the presence of newly formed elastic fibrils. In the tail the fibrous scar-tissue is no more recent than that in the remainder of the gland; it appears indeed to be of somewhat earlier origin. But in the tail active inflammation is demonstrated by extreme cellular infiltration and by the accumulation of iron-pigment in the remnants of parenchyma; in the remainder of the gland there is very little infiltration. The cytology of the infiltration suggests a very chronic pyogenic infection. The endophlebitis and endarteritis cannot be

accepted as proof of syphilitic infection. The coli-form bacilli detected in the tail may have been responsible for the inflammation, but a very few diplococci were also found. There is no evidence in the sections that the infection reached the gland through its ducts. My experience would lead me to expect that so great a destruction of the pancreas was associated with a persistent, spontaneous glycosuria.

I am indebted to Dr. William Morris for the drawing of the mid-brain and medulla, and to Mr. J. R. Ford for the drawings of the microscopic preparations.

IS ARTERIAL HYPERTENSION COMPENSATORY AND CONSERVATIVE?

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THERE are many clinicians to-day who consider arterial hypertension as compensatory and conservative, a sort of "Nolle me tangere," and who believe that any efforts addressed to the lowering of excessive blood-pressure are fraught with dire results to heart and kidneys in this class of cases. In our observation, however, of the subjects of hypertension, both in the Internal Medical service at Barnes Hospital, and in private practice we have for some time been impressed with the number of compensated and decompensated cases of hypertension, in which the cardiac and renal compensation was not disturbed, and decompensation in these organs improved or restored through a progressive lowering of blood-pressure extending over a period of time, sufficiently long to establish the blood-pressure fall as more or less permanent. And it is a group of cases as just described, presented on a purely clinical basis that we are desirous to have speak for themselves in reply to the query in our title.

A few brief historical allusions, however, may also assist in the further elucidation of the question. Traube¹ it was, who, restricting his study solely to cases of chronic Bright's disease, first attributed rise of blood-pressure to a vaso-constriction of the renal vessels. Ewald and Potain, however, argued for a more general hypertension due either to a vaso-constriction of the whole arterial tree from the presence in the blood of some toxic so-called pressor substance, or to a reflex, general vaso-constriction from a renal irritation. Mabomed² the

brilliant young clinician at Guy's Hospital, who, believing that the significance of high blood-pressure was profound and its effects far reaching introduced the term, "prealbuminuric stage of Bright's disease;" holding that before Bright's disease manifested itself there was a period of high blood-pressure during which the encroachment of the disease could be foretold, and perhaps forestalled. And with these views Riegel³ agreed. Clifford Allbutt⁴, however, found that many cases of prealbuminuric Bright's disease never actually developed Bright's disease, and one of his cases of hypertension lived eighteen years without ever showing any symptoms or signs of a renal lesion; her death having been finally due to a cerebral hemorrhage. He, with Mabomed, von Basch and Huchard, recognized that rising blood-pressure did not necessarily mean an incipient stage of Bright's disease. Before Clifford Allbutt's statement that many cases of hypertension never developed nephritis, Lawrence⁵ found that clinical reports were by no means unanimous in declaring hypertension to be compensatory and conservative, and quotes numerous investigators to the effect that a lowering of pressure was coincident with improvement.

Experimental studies also show, first, that decreased renal tissue is not the cause of hypertension; second, that hypertension does not cause increased flow of blood through the kidney; third, that hypertension is not accountable for polyuria. Janeway⁶ in 458 patients with a systolic pressure of 160 or over, found 116 with neither albumin nor casts, and 9 cases with albumin but no casts, and vice versa. He further states that there is undoubtedly a large group of cases in which the hypertension and arteriosclerosis are the primary conditions, the sclerotic process extending later to the renal vessels, developing the arteriosclerotic kidney as a secondary condition. Moschowitz⁷ says, "Hypertension may occur with normal or practically normal kidneys and that evidence is adduced to indicate that in many instances, at least the pathological changes in the kidneys in Bright's disease, are the result rather than the cause of hypertension; such a conception would render it very probable that the hypothesis of the primary vascular origin of the lesion of Bright's disease is correct. Arteriosclerosis and Bright's disease have therefore the same pathogenesis, the lesion in each being modified by the nature of the organ. Furthermore all available evidence at our command seems to show that arterio-

sclerosis is the result of the hypertension; not the cause that brought about the hypertension. Every persistent hypertension, unless complicating factors are introduced, will eventually result in the graver evidences of decompensation, represented by the clinical picture of cardiac insufficiency." (Also renal insufficiency may be correctly added in many cases.)

On the other hand, Ringer⁸ says, "Personally I am inclined to consider every case of hypertension as a potential case of chronic nephritis, just as I am inclined to consider every case of nephritis a potential case of uræmia." If by this statement the author means to convey that every case of hypertension is a potential case of nephritis in the sense that sooner or later the sclerosis resulting from the hypertension will involve the renal arteries and its branches and thereby result in the arteriosclerotic kidney, we then heartily agree, although reservation must even here be made for certain exceptional cases; but if on the other hand, he means to imply that with no signs at all of nephritis, still the condition must have started with a nephritis existing without any tangible evidence of its existence, simply because hypertension must have nephritis as its cause, then we must most positively disagree with such an assumption, for we do not think it logical to assume the existence of a disease without the slightest tangible evidence of the presence of such a disease. Allen⁹ in his most valuable contribution on salt and water retention as causes of hypertension writes as follows: "But any practitioner who will conform his treatment to one definite condition, *viz.*, the necessity of the organism to force a filtrate of dissolved substances through advanced and partially blocked glandular filter can readily demonstrate for himself the compensatory element in the hypertension by observing the fall in pressure and the relief of some attendant symptoms, when proper diet reduces the quantity of the filtrate to a minimum."

But is hypertension compensatory even under the conditions just described? On the contrary is it not just as plausible to attribute the hypertension to an accumulation in the blood of some pressor substances not necessarily the result of faulty renal elimination primarily, but possibly due to excessive intake of such vaso-constricting bodies and the vaso-constricting effect of such bodies on the arterial tree involving naturally the renal artery and its branches, thus diminishing the blood supply to the kidney, and thereby reducing the

urinary secretion? How can we expect hypertension to force substances through the kidney when the renal arteries and their branches with all the rest of the arterial tree, are in a state of marked vaso-constriction?

Remove the pressor substances from the blood, then vaso-constriction gradually gives way to vaso-dilation, the arterial tree together with the renal vessels dilate, blood-pressure falls, more blood reaches in a given time the now dilated renal vessels, and the kidneys in compliance with the law governing all secretory glands pour out more secretion as the blood reaching these organs is increased, but the increased secretion results not from the hypertension, but from a lowering of the hypertension.

In this connection Howell¹⁰ states that "conditions such as asphyxia, strychnine poisoning or painful stimulation of sensory nerves, which cause a general vaso-constriction, tend to diminish the blood flow through the kidney." Also, "any change which will increase the difference in pressure between the blood in the renal artery and renal vein will tend to augment the flow of blood unless antagonized by a simultaneous constriction in the smaller arteries of the kidney itself." But in arterial hypertension we have pressor substances which cause a general vaso-constriction involving of course the renal arteries and their branches in a condition of simultaneous constriction—so that we have both conditions present which Howell enumerates as tending to diminish rather than increase kidney function. Moreover, if arterial hypertension be compensatory in nephritis, why is it that in at least one large class of the nephritides classified by Volhard and Fahr¹¹ as degenerative nephritis, these authors state, hypertension and cardiac hypertrophy never obtain; although marked retention of salts and water together with large amounts of albumin and numerous casts are the rule in this class of cases?

Also how is to be explained a greatly diminished urinary output almost amounting to suppression in some cases of very high arterial pressure, even before any definite evidence of cardiac decompensation appears? In renal cases where the arterial hypertension results from a nephritis primarily, even in such cases we do not consider the hypertension as really in any way compensatory, for the hypertension is simply the result of the accumulation of pressor substances in the blood due largely to faulty renal function and causing vaso-constriction

of the arterial tree and thereby hypertension; the latter representing the effect of the action of the pressor substances on the vaso-constricting apparatus of the arteries, and not therefore developed for the purpose of driving more blood through damaged kidneys for as we have just stated, we have cases of extensive nephritis and marked retention of certain substances in the blood from faulty renal function and yet no hypertension at all and on the other hand we have cases of very marked hypertension without any tangible evidence of nephritis. If therefore, arterial hypertension be not compensatory the old idea of efforts at its reduction being fraught with dire disaster to kidneys and heart is not tenable, a view which our reported cases will, we think, help to make clear. If the arterial hypertension be not compensatory how then can it be conservative?

The answer to this question requires some consideration of arteriosclerosis. Allbutt divided arteriosclerosis into three main divisions:

- (1) Those associated with Bright's disease;
- (2) Those not associated with Bright's disease but always accompanied by high blood-pressure, called hyperpiesis;
- (3) Those not associated with Bright's disease or high blood-pressure, and called senile or involutionary.

In group one, the cause of the arteriosclerosis is either the hypertension or certain retained toxic substances due to deficient renal function. In the third group the cause is a degenerative process due either to age or hereditary weakness in the arterial system. In the second group the cause is solely the hypertension.

According to Adami and Nichols¹² "Arterial contraction and particularly a generalized arterial contraction is the primary cause of heightened blood-pressure, and the persistent rise of blood-pressure, or the condition termed by Sir Clifford Allbutt a hyperpiesis, is the commonest precursor of arteriosclerosis."

Clifford Allbutt¹³ says: "That high pressures and friction are competent to set up arteriosclerosis, was clearly shown by Roy and Adami, and I have adduced familiar proof of it in the alteration of the arterial wall at critical points, as at bifurcations, at narrows, normal or morbid, and again at dilatations with change of wave leading to distortion and elongation," and again, "vessels which are incessantly subjected to hydrostatic stresses cannot but betray these effects, and these the more as by one coöperating frailty or another,

their coats may be less fixed to withstand them; but by arteriosclerosis of high pressure properly so-called, we mean surely lesions primarily and mainly thus produced in vessels primarily sound.

Arterial hypertension therefore is a potent cause of arteriosclerosis and we have but to consider for a moment the effects of the arteriosclerosis on the cardio-vascular renal apparatus to realize that high blood-pressure is far from being in any way conservative.

In the heart, therefore, there results from such a strain and from sclerosis of the coronary arteries, angina pectoris, chronic myocarditis, chronic cardiac dilation and cardiac decompensation. In the brain, intermittent cerebral claudication, thrombosis of cerebral vessels and apoplexy. In the aorta, thoracic aneurism, and in the kidneys the arteriosclerotic kidney.

These of course are only the most marked effects of arteriosclerosis, sufficient, however, to stamp it as a condition, not in any sense conservative but always absolutely destructive sooner or later to heart, blood-vessels and kidneys and a most frequent cause of death, especially in individuals at, or past middle life. Some clinicians remark, why, if cases of hypertension may go on for years in a state of comfort why disturb their equanimity of mind through calling blood-pressure to their attention? Allbutt then replies, "Young and sound vessels may resist this degeneration for years, but surely it comes about even in them; speaking generally, then a thickened artery means plus stress or minus elastic value or an algebraic sum of both in various proportion." And why submit the many to so dangerous a strain which only the lucky few may for time bear in safety? Especially if it be possible to protect a goodly proportion of the many from the ravages of so serious and fatal a malady for longer or shorter periods of time.

That reduction of blood-pressure is not fraught with dire results to heart and kidneys in many cases is shown in the accompanying charts and tables. These cases of arterial hypertension have come under close observation either in the Internal Medical Service of Barnes Hospital or in private practice, and have been classified as follows:

- (1) As hospital cases;
- (2) As out-patients or ambulatory cases.

Each of these two divisions has been subdivided into those that are compensated and into those that are decompensated. That is,

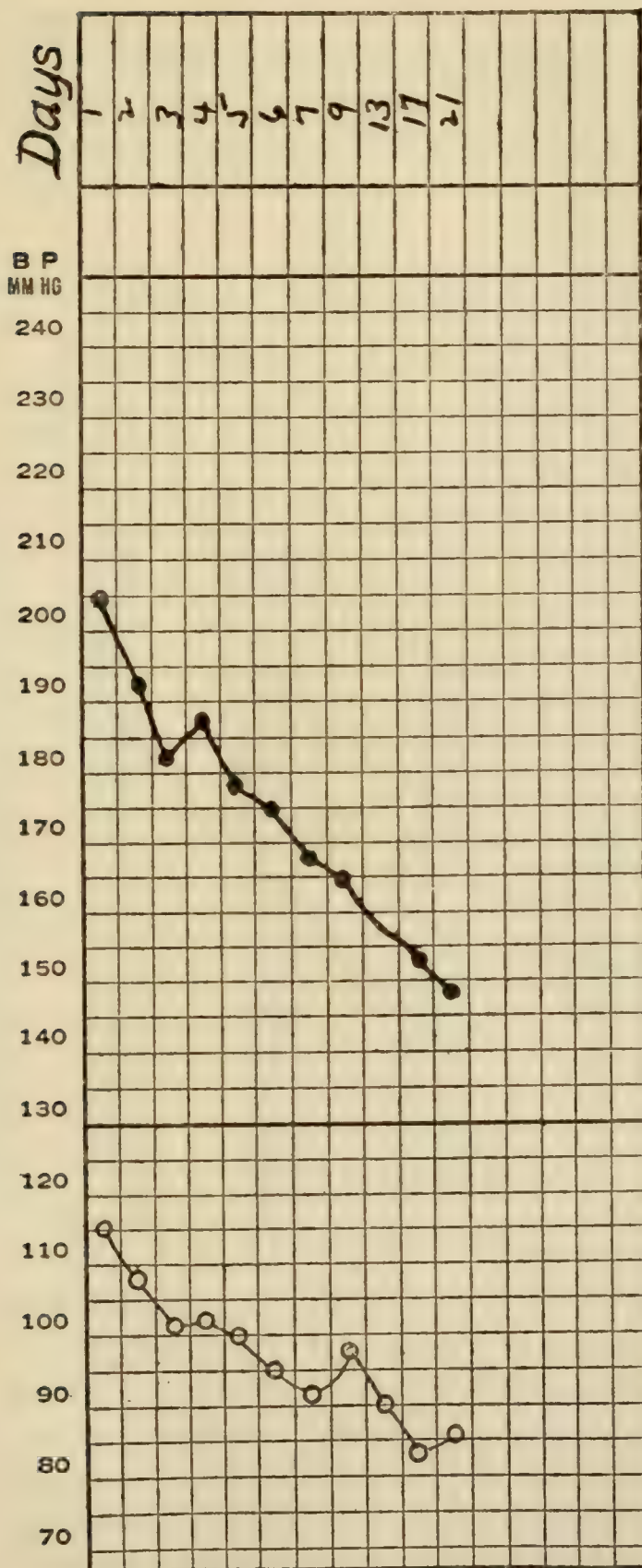
the compensated cases are those which have so far weathered the strain of the hypertension on heart, arteries and kidneys, without as yet giving any evidence of disturbed function in these organs; arteriosclerosis not having as yet developed to any appreciable degree, while on the other hand the decompensated cases are those showing various abnormalities of function in the cardio-vascular apparatus due to the effects of long continued high blood-pressure and resulting arteriosclerosis. The compensated class of cases came under observation for relief of ailments not directly involving the cardio-vascular renal system.

Charts *I* and *III* show the definite gradual, though constant fall in blood-pressure in the hospital compensated and decompensated groups respectively, while charts *II* and *IV* present the same phenomena in the out-patient compensated and decompensated groups.

These blood-pressure curves were obtained by averaging the blood-pressure observations of all the cases in each hospital group daily for the first week and every other day during the second and third weeks, and in the out-patient groups the blood-pressure curves were figured by averaging weekly observations, thus obviating the objection of the variations in blood-pressure observations, for we have here groups of cases showing definite reductions in the blood-pressure at dates sufficiently close, yet during periods of time sufficiently long to establish the results as permanent, provided the conditions under which the patients live remain constant. And yet, in cases appearing in charts *I* and *III* there was not the slightest evidence of disturbance of the compensatory balance either in heart or kidneys as a result of the definite fall in blood-pressure, while amongst the cases comprised in charts *II* and *IV* not only was there not the least manifestation of any increase in the cardiac and renal decompensation consequent on an equally definite and permanent average drop of blood-pressure, but on the contrary either a definite improvement in cardiac and renal decompensation, or even in many cases an actual restoration of compensation in these organs.

Charts *V* and *VI* illustrate typical reductions in blood-pressure in individual cases of compensated and decompensated arterial hypertension respectively.

CHART No. I.



Showing average blood-pressure in Group I of hospital cases with cardiac compensation. Here we see a group of 25 hospital cases in which complete heart compensation was not in the least disturbed with definite constant fall in blood-pressure covering a period of three weeks.

Table I presents the urinary findings in hospital and out-patient compensated groups, demonstrating no disturbance in the renal organs as a result of the blood-pressure fall, while table II speaks for an actual improvement in the condition of the kidneys consequent on lowering of blood-pressure in the decompensated hospital and ambulatory groups.

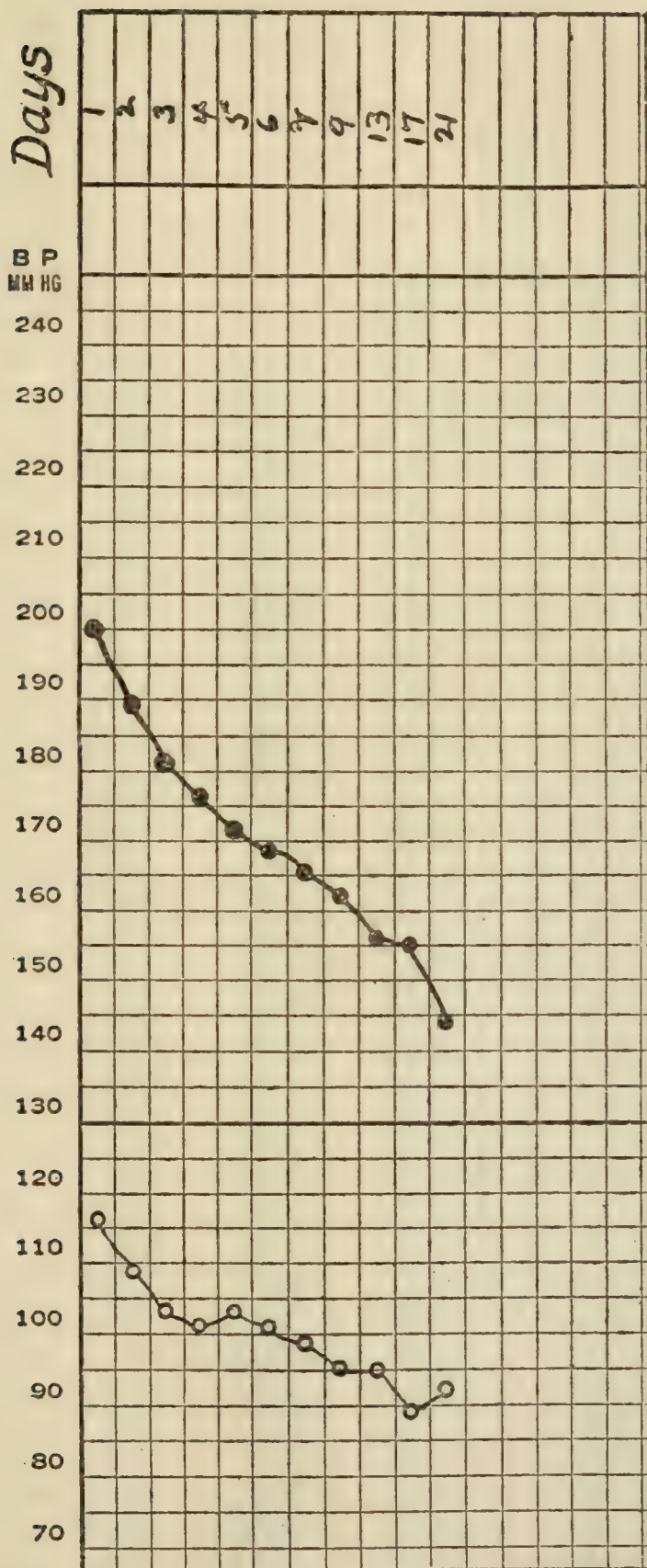
TABLE I
Shows No Disturbance of Renal Function from Lowering of Blood-pressure.

No.	Age	Blood-pressure		P. S. P.		Urine				Days Observed
		Entrance	Discharge	Entrance	Discharge	Entrance		Discharge		
1	48	205-110	150- 75	68		Alb +	Cast +	Alb +	Cast +	23
2	58	220-128	150- 95	57		Alb +	Cast0	Alb0	Cast0	28
3	61	220-105	135- 85			Alb +	Cast0	Alb0	Cast0	50
4	56	185-114	155- 90	43		Alb0	Cast0	Alb0	Cast0	28
5	62	170- 80	150- 70	40		Alb +	Cast +	Alb +	Cast +	41
6	49	190-120	165-105	27		Alb + + +	Cast + +	Alb + + +	Cast + +	15
7	63	180-100	155- 85	74		Alb0	Cast0	Alb0	Cast0	11
8	72	220-128	132- 70	48	49	Alb +	Cast +	Alb0	Cast0	50
9	60	220-150	170- 85	50		Alb0	Cast0	Alb0	Cast0	12
10	50	212-128	160-100	15	25	Alb + + + +	Cast + + +	Alb + +	Cast +	30
11	61	240-110	200- 98	40		Alb + + + +	Cast + +	Alb + +	Cast +	14
12	63	180-120	120- 95	55		Alb +	Cast +	Alb0	Cast0	21
13	44	170- 50	152- 56	63		Alb0	Cast0	Alb0	Cast0	7
14	60	173-100	154- 73	58	74	Alb +	Cast0	Alb0	Cast0	18
15	53	220-140	135- 90	75		Alb +	Cast0	Alb0	Cast0	14
16	53	212-124	155- 80	32		Alb0	Cast0	Alb0	Cast0	30
17	47	212-123	162- 92	40	42	Alb +	Cast +	Alb0	Cast0	9
18	65	240-120	145- 80	30		Alb0	Cast +	Alb0	Cast0	25
19	49	233-133	160-117	67		Alb +	Cast0	Alb0	Cast0	10
20	61	190-120	160-115	50	55	Alb +	Cast0	Alb0	Cast0	90
21	32	210-130	155-100			Alb0	Cast0	Alb0	Cast0	43
22	62	275-165	148- 98			Alb +	Cast0	Alb0	Cast0	375
23	55	300-140	175-100			Alb + + + +	Cast + + +	Alb +	Cast +	53
24	55	185-105	125- 90			Alb0	Cast0	Alb0	Cast0	425
25	58	190-115	160- 90			Alb0	Cast0	Alb0	Cast0	392
26	62	240-100	155-100			Alb0	Cast0	Alb0	Cast0	660
27	61	210-110	160-105			Alb0	Cast0	Alb0	Cast0	120
28	55	185- 95	148- 80			Alb +	Cast0	Alb0	Cast0	90
29	58	180- 90	145- 80			Alb0	Cast0	Alb0	Cast0	90
30	57	190- 85	140- 73			Alb0	Cast0	Alb0	Cast0	110
31	55	180- 90	135- 80			Alb0	Cast0	Alb0	Cast0	31
32	48	185- 90	125- 80			Alb0	Cast0	Alb0	Cast0	47
33	50	178- 90	115- 75			Alb +	Cast0	Alb0	Cast0	38

This table shows no increase in kidney disturbance as represented by an increase in amount of albumin or casts, or by a decrease in the P. S. P. determination in hospital and ambulatory compensated groups.

To obtain results such as these however, cases of arterial hypertension must be recognized and treated at a period sufficiently early to anticipate the incurable conditions established primarily in the arterial tree, secondarily in heart and kidney by the arteriosclerotic process, for even in our decompensated groups of cases herein presented, the arteriosclerotic process had not progressed sufficiently to rob the arterial tree of its elasticity, or, as we say, of its vasomotor response; and hence when the vaso-constricting or so-called pressor

CHART No. II



Showing average blood-pressure curve in Group II of hospital cases with cardiac compensation. Here we see a group of 38 hospital cases in which the heart decompensation was not aggravated, but on the contrary greatly improved and in many cases restored with an equally definite constant fall in blood-pressure.

TABLE II

Instead of Showing Any Increase in Renal Decompensation this Table Shows an Actual Improvement in Kidney Function with a Reduction in Blood-pressure.

No.	Age	Blood-pressure		P. S. P.		Urine				Days Observed
		Entrance	Discharge	Entrance	Discharge	Entrance		Discharge		
1	47	162- 82	100- 55	40		Alb + + + +	Cast + +	Alb + +	Cast +	43
2	40	190-120	117- 75	25	30	Alb + +	Cast +	Alb +	Cast 0	21
3	48	268-198	195- 90	10	26	Alb + + +	Cast + +	Alb + +	Cast +	41
4	58	190- 95	134- 70	72		Alb +	Cast +	Alb +	Cast +	11
5	50	186-136	160-130	40	35	Alb +	Cast +	Alb +	Cast +	26
6	53	163-114	135-104	22		Alb +	Cast +	Alb +	Cast +	42
7	45	163-103	122- 72	33	39	Alb 0	Cast 0	Alb 0	Cast 0	14
8	65	190-110	126- 82	23	24	Alb + +	Cast +	Alb + +	Cast +	30
9	48	268-198	180-100	10	26	Alb + + + +	Cast + + + +	Alb + +	Cast + +	43
10	42	200-110	160-105	55	65	Alb +	Cast +	Alb 0	Cast 0	35
11	51	192-100	143- 90	22	23	Alb 0	Cast 0	Alb 0	Cast 0	20
12	50	172- 80	125- 70	53	43	Alb +	Cast +	Alb +	Cast +	29
13	74	230-100	168- 80	35		Alb +	Cast 0	Alb +	Cast 0	10
14	48	183- 77	123- 50	50		Alb + + +	Cast + +	Alb 0	Cast 0	25
15	56	180-100	120- 75	71		Alb +	Cast +	Alb 0	Cast 0	54
16	54	205-120	160- 95	45	55	Alb + +	Cast 0	Alb 0	Cast 0	33
17	52	205- 90	145- 70	60		Alb + +	Cast +	Alb 0	Cast 0	30
18	65	215-120	170-110	56		Alb +	Cast +	Alb +	Cast 0	14
19	50	205-145	175-125	45	54	Alb + +	Cast + +	Alb +	Cast +	41
20	58	170-130	145- 90	10	17	Alb + +	Cast + +	Alb + +	Cast +	48
21	62	180-110	152- 92	45	48	Alb +	Cast +	Alb 0	Cast 0	16
22	28	250-140	210-130	42		Alb +	Cast +	Alb +	Cast 0	20
23	47	200-140	175-120	28	45	Alb + + + +	Cast + + + +	Alb +	Cast 0	50
24	45	220-175	186-145	83		Alb +	Cast 0	Alb 0	Cast 0	34
25	60	174- 85	130- 70	61		Alb +	Cast 0	Alb 0	Cast 0	31
26	59	208-130	145-110	51		Alb +	Cast +	Alb 0	Cast 0	26
27	44	195- 85	150- 60	42		Alb + + +	Cast + + +	Alb 0	Cast 0	30
28	73	195-100	140- 70	34	30	Alb 0	Cast 0	Alb 0	Cast 0	27
29	51	200-105	155- 80	41		Alb 0	Cast 0	Alb 0	Cast 0	21
30	50	180-125	125- 90	15	28	Alb 0	Cast 0	Alb 0	Cast 0	24
31	59	210-110	132-102	67		Alb + +	Cast + +	Alb +	Cast +	19
32	65	215-105	135- 85	65		Alb +	Cast +	Alb 0	Cast 0	14
33	45	167-102	138- 82	32	39	Alb 0	Cast 0	Alb 0	Cast 0	14
34	60	186- 90	158- 75	48		Alb + +	Cast +	Alb + +	Cast +	21
35	56	235-120	150- 90	43	40	Alb + +	Cast +	Alb +	Cast 0	60
36	57	230-150	170-105	40		Alb +	Cast 0	Alb +	Cast 0	15
37	55	210-140	160-110	38	45	Alb +	Cast +	Alb +	Cast 0	20
38	60	185- 85	155- 75			Alb +	Cast 0	Alb +	Cast 0	24
39	58	195-100	155- 80			Alb 0	Cast 0	Alb 0	Cast 0	18
40	56	200-105	150- 80			Alb +	Cast +	Alb 0	Cast 0	21
41	40	185-135	145-110	40		Alb + + + +	Cast + +	Alb + +	Cast +	360
42	64	210-110	140- 80	55		Alb + +	Cast + +	Alb +	Cast +	365
43	60	220-100	165-105	60	60	Alb 0	Cast 0	Alb 0	Cast 0	130
44	71	248-120	175-130	40	40	Alb +	Cast +	Alb +	Cast +	138
45	64	200- 90	150- 75	60	65	Alb 0	Cast 0	Alb 0	Cast 0	482
46	52	170- 90	150- 80	60	60	Alb +	Cast 0	Alb +	Cast 0	150
47	52	200-110	170- 95	40	45	Alb +	Cast +	Alb +	Cast +	600
48	66	240-115	155- 90	60	60	Alb 0	Cast 0	Alb 0	Cast 0	620
49	69	228-140	175-130	50	50	Alb +	Cast 0	Alb +	Cast 0	300
50	52	185-125	135- 90	60	61	Alb 0	Cast 0	Alb 0	Cast 0	64
51	60	240-140	180-120	40	45	Alb +	Cast +	Alb +	Cast +	240
52	61	208-120	170-130	65	65	Alb 0	Cast 0	Alb 0	Cast 0	100
53	59	190-110	160- 95			Alb + +	Cast +	Alb +	Cast 0	61
54	45	230-120	185-100			Alb + + + +	Cast + + +	Alb + +	Cast +	137
55	68	230-120	175-110			Alb + +	Cast +	Alb +	Cast +	18
56	59	210-130	170-100			Alb 0	Cast 0	Alb 0	Cast 0	256
57	64	180- 80	130- 80			Alb + +	Cast +	Alb +	Cast 0	72
58	62	180-100	135- 80			Alb + +	Cast + +	Alb + +	Cast +	120
59	54	190-105	140- 80			Alb +	Cast +	Alb +	Cast +	80
60	52	220-130	165- 90			Alb + +	Cast +	Alb +	Cast 0	75
61	40	204-108	150- 85			Alb + + +	Cast 0	Alb 0	Cast 0	760
62	54	215-130	170-105			Alb + +	Cast +	Alb +	Cast 0	101
63	65	190-120	155- 95			Alb + +	Cast +	Alb 0	Cast 0	730
64	59	225-120	168- 88			Alb 0	Cast 0	Alb 0	Cast 0	50
65	48	205-120	150- 95			Alb +	Cast +	Alb 0	Cast 0	74
66	51	210-110	160- 90			Alb + +	Cast +	Alb +	Cast +	80
67	51	220-115	160- 85			Alb + +	Cast 0	Alb +	Cast 0	75

This table shows a decreased amount of albumin and casts in 37 cases, a stationary condition in 30 cases and an increased amount of albumin and casts in no cases at all. It also shows an increased per cent. in P. S. P. in 19 cases, a stationary per cent. in 6 cases and a lowered per cent. in only 4 cases of hospital and ambulatory decompensated groups.

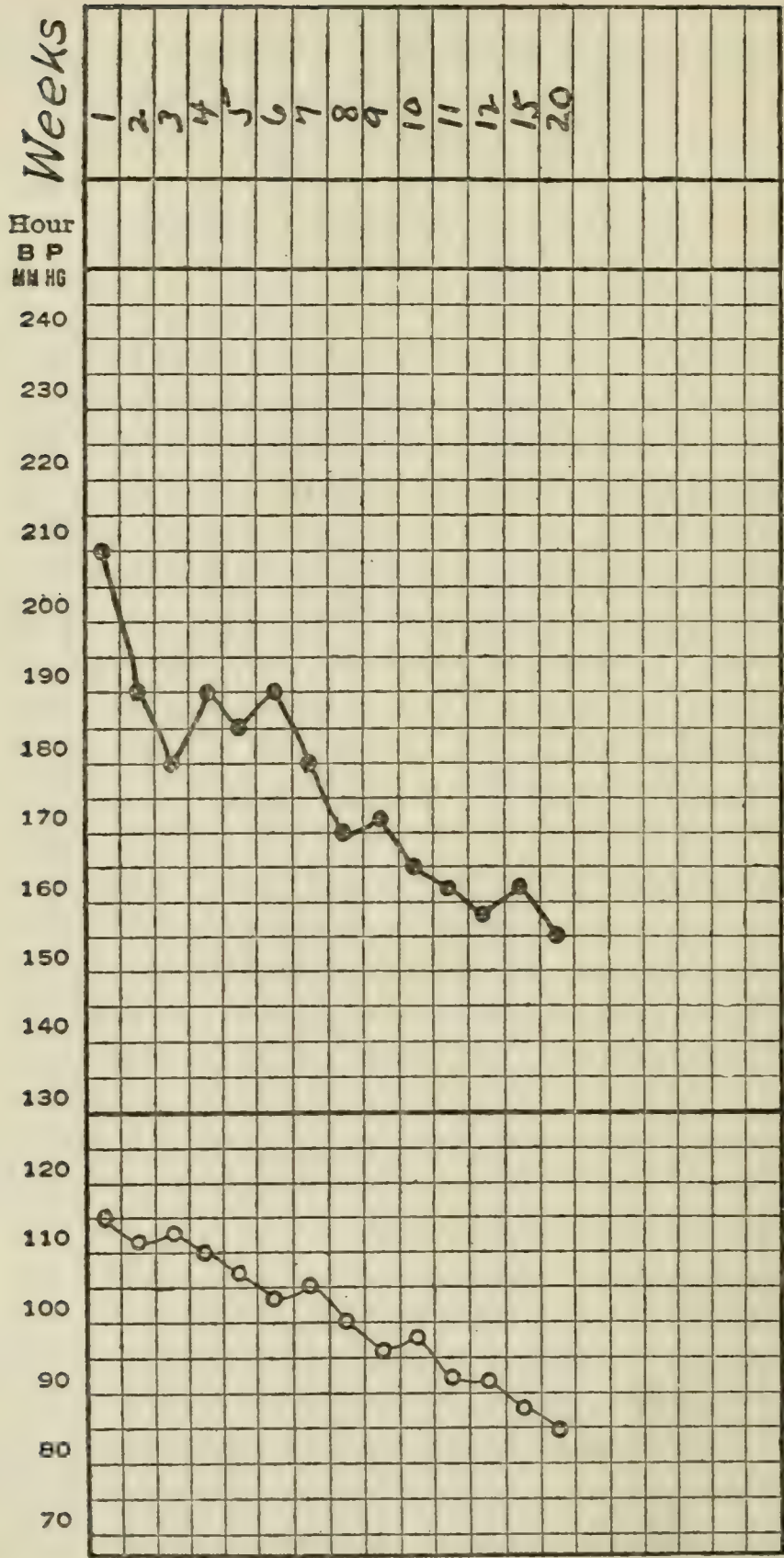
substances were removed from the blood, the arterial tree could dilate and thus lower blood-pressure; whenever on the contrary the arteriosclerotic process advances to the point of fixation of the arterial tree as a rigid set of tubes, the vasomotor property of dilation is lost and blood-pressure can no longer be lowered through vaso-dilation and the only remaining way of blood-pressure reduction in these advanced cases is that of progressive weakening of the cardiac pump back of the persistent constant strain of hypertension until heart-failure and death close the scene.

We have, however, so far outlined three distinct groups of cases: (1) The compensated; (2) the decompensated both with elasticity of arterial tree or vasomotor response still retained; (3) the decompensated group with the vasomotor response lost. And to these, two others are to be added, the first of which is a group of patients who have been endowed by nature with such a wonderful cardio-vascular renal apparatus, as to apparently withstand for an almost indefinite time the strain of arterial hypertension, even after the vasomotor response has been lost rendering thereby the hypertension irreducible. This is the so-called benign form as described by Stengel and others. These cases are, however, the few fortunate ones, and, as we have said, are not to be by any means considered the rule. The second group to be added is where the heart muscle shows such remarkable power of "come-back" that in spite of breaking down behind a long continued irreducible hypertension, the blood-pressure only falling as a result of the failing cardiac pump, this same crippled pump will under the influence of rest and proper treatment so far regain its propelling force as to raise the blood-pressure again to the level of hypertension present at the time of the break in cardiac compensation and carry on again effectively the circulation under the old conditions of hypertension for a longer or shorter period of time.

Based on these views the senior writer has formulated the following classification of arterial hypertension and its results:

(1) *Vasomotor response retained, blood-pressure lowered by vaso-dilation; cardiac and renal compensation conserved.*—Here we not only find no disturbance in either cardiac or renal compensation, but, with what must prove to be actual conservation of the cardiac and renal compensation, as also of the walls of the arterial tree and cardiac muscle with a reduction in blood-pressure.

CHART No. III



Showing average blood-pressure curve in Group III, the ambulatory cases with cardiac compensation. Here we have a group of 8 ambulatory cases in which the heart compensation was not in the least disturbed with a definite fall in blood-pressure.

(2) *Vasomotor response retained, blood-pressure lowered by vasodilation, cardiac and renal decompensation improved.*—With the lowering of the blood-pressure the strain on the struggling heart is greatly lessened giving the fatigued cardiac muscles an opportunity to regain their tone with a resulting marked improvement, and in many cases complete restoration in the cardiac compensation and of renal decompensation, secondary to the weakened heart.

(3) *Vasomotor response refractory, blood-pressure is irreducible, cardiac and renal compensation maintained by integrity of cardiac muscle.*—Here the heart is sufficiently strong to maintain the cardiac and renal compensation for a longer or shorter time, in spite of increased strain thrown on it by a heightened blood-pressure. Just so long as the heart thus remains compensated do these cases continue symptomless.

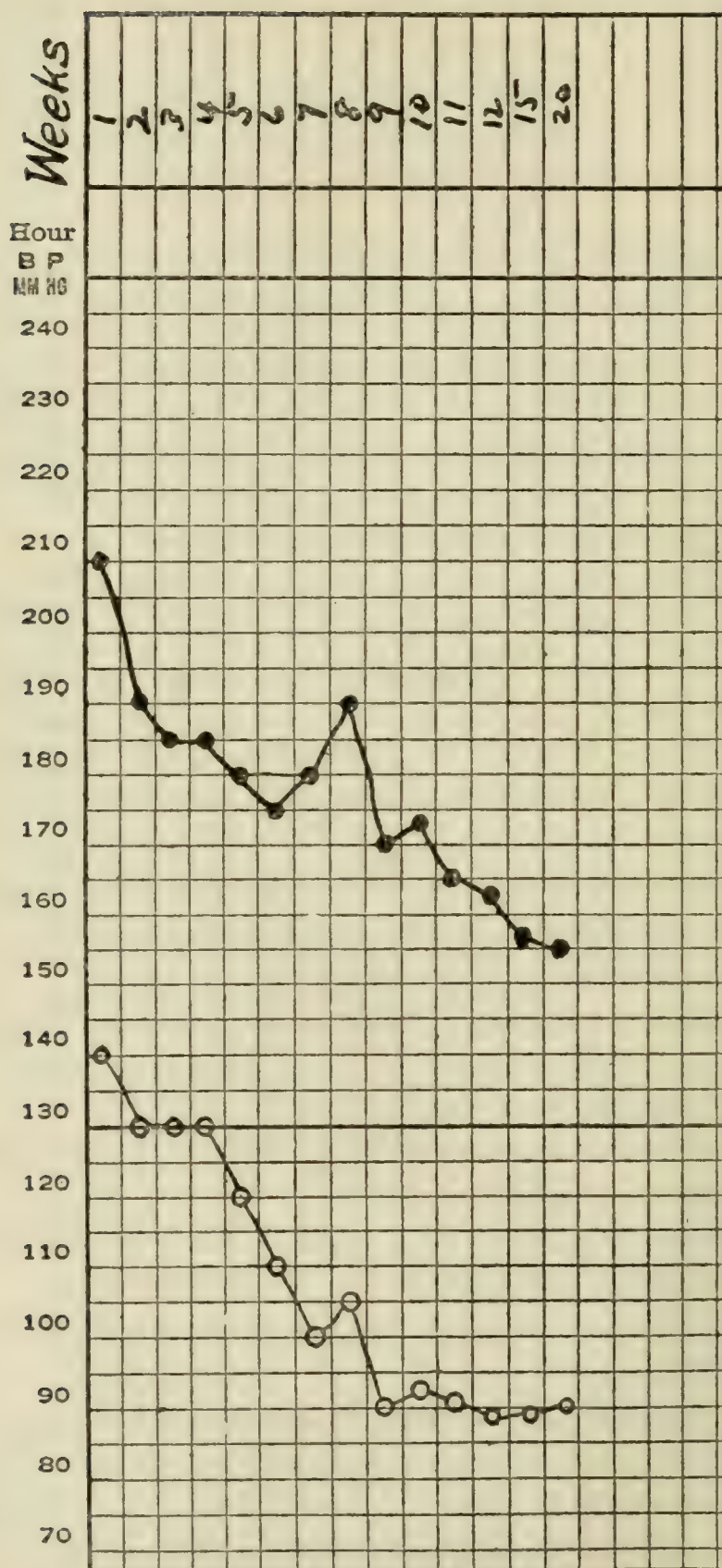
(4) *Vasomotor response refractory, blood-pressure raised through increased tone of cardiac muscle with improvement in cardiac and renal decompensation.*—With hypertension still irreducible, a resulting break in cardiac and renal compensation has at last occurred, but the heart through rest or other treatment has so regained its tone as to raise the blood-pressure to the point necessary for compensation, with a more or less permanent relief in symptoms. The hypertension however, has not been conservative and only necessary for compensation because of the loss of vasomotor response, and compensation under such a strain cannot be long maintained by the heart.

(5) *Vasomotor response refractory, the cardiac and consequent renal decompensation irremediable and death the ultimate result in one of the three following ways:*

- (a) Hypertension progressively lowered with gradual cardiac failure and death.
- (b) Hypertension unchanged, sudden death is frequent from acute cardiac dilatation.
- (c) Hypertension raised, sudden death from acute cardiac dilatation is the rule.

With the hypertension persistently irreducible this group of cases furnishes the sequence to the majority of those in groups 3 and 4. The pumping force of the heart becomes permanently crippled with a progressive increase in all the symptoms of both cardiac and renal decompensation, on to the fatal termination; for in this class of

CHART No. IV.



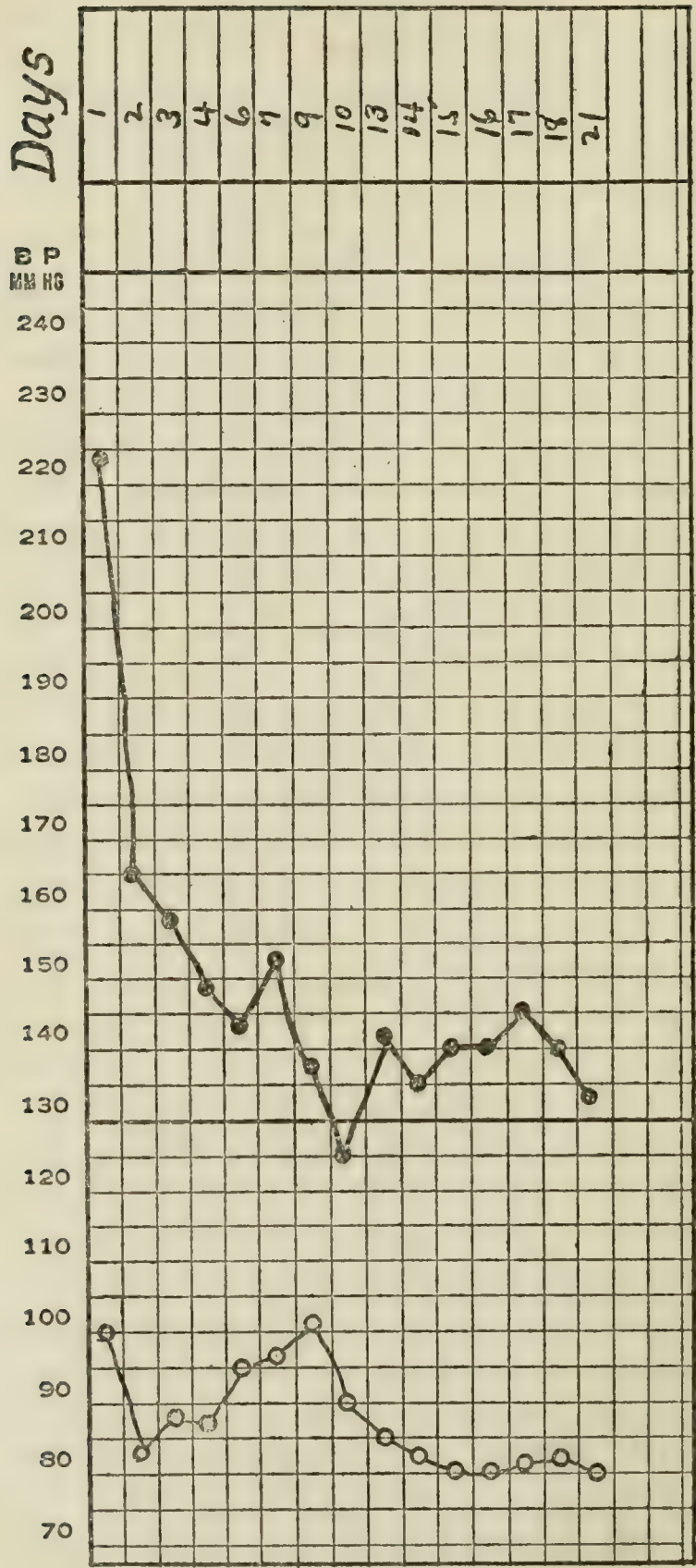
Showing average blood-pressure curve in group IV, the ambulatory cases with cardiac compensation. Here also in this group of 29 ambulatory cases the decompensation was not only not aggravated but on the contrary greatly improved and in many cases actually restored with equally definite constant fall in blood-pressure.

cases the reserve cardiac energy becomes exhausted and the organ is unable longer to respond to remediable agencies. It is these final hopeless results in hypertension cases as occur in this group which should so strongly plead for early control of high blood-pressure, before the organic changes occur in blood-vessels, heart and kidneys, that ultimately result in such disaster.

It must now be apparent then that the heart is under as great if not greater strain in arterial hypertension, than it is in valvular disease, greater, we think because after hypertension results in arteriosclerosis, which in its turn causes loss of elasticity and vasomotor response, the heart not only is obliged to pump against the high peripheral resistance but is moreover robbed of the great assistance of the elastic recoil of the aorta and entire arterial tree, during the diastole of the organ whereby according to Howell¹⁴—"As much blood is forced on in the circulation as is driven out of the organ during each systole." And if we were to ask, that if it were possible what would be logically the first indication in the treatment of a case of valvular disease, the reply should be prompt—remove the cause by replacing the diseased valves with new ones, as we would repair any pump in mechanics. Now this of course, unfortunately, can never be done, but on the contrary the strain on the heart in arterial hypertension can be removed by lowering the arterial pressure at a time sufficiently early in the progress of the case to anticipate, and thereby prevent, or at least delay for a long period of time, arteriosclerosis and its end results—cardiac and renal insufficiency. And since reduction of arterial pressure wherever possible it is not fraught with dire results, but on the contrary with most beneficial and life-saving effects, to accomplish such blood-pressure reductions would seem therefore to be not only most salutary but actually our bounden duty that we may thereby conserve heart, arteries and kidneys in a malady tending progressively toward a fatal termination, if not so controlled. It should be impressed therefore on the patient the importance of having his blood-pressure watched at least from the beginning of the fourth decade on, just as he is advised to have teeth and urine examined; that in this way arterial hypertension may be detected early when control of the affection is easier and more effective, for the prevention of the late disasters to heart, arteries and kidneys.

The subject of the causes of hypertension as also its treatment

CHART No. V



Showing blood-pressure curve in a typical case of Group I.

have been intentionally omitted as we were concerned only with the question as to whether or not high blood-pressure was compensatory and conservative. Suffice it to say that the reductions in blood-pressure in the cases reported were obtained mainly through rest, diet, elimination, removal of focal infections, etc., and not through drugs of the vaso-dilator class. The blood-pressure observations moreover were all made with mercury instruments and as far as possible at about the same time in the morning.

CONCLUSIONS

(1) Arterial hypertension does not exert a compensatory influence over the renal function.

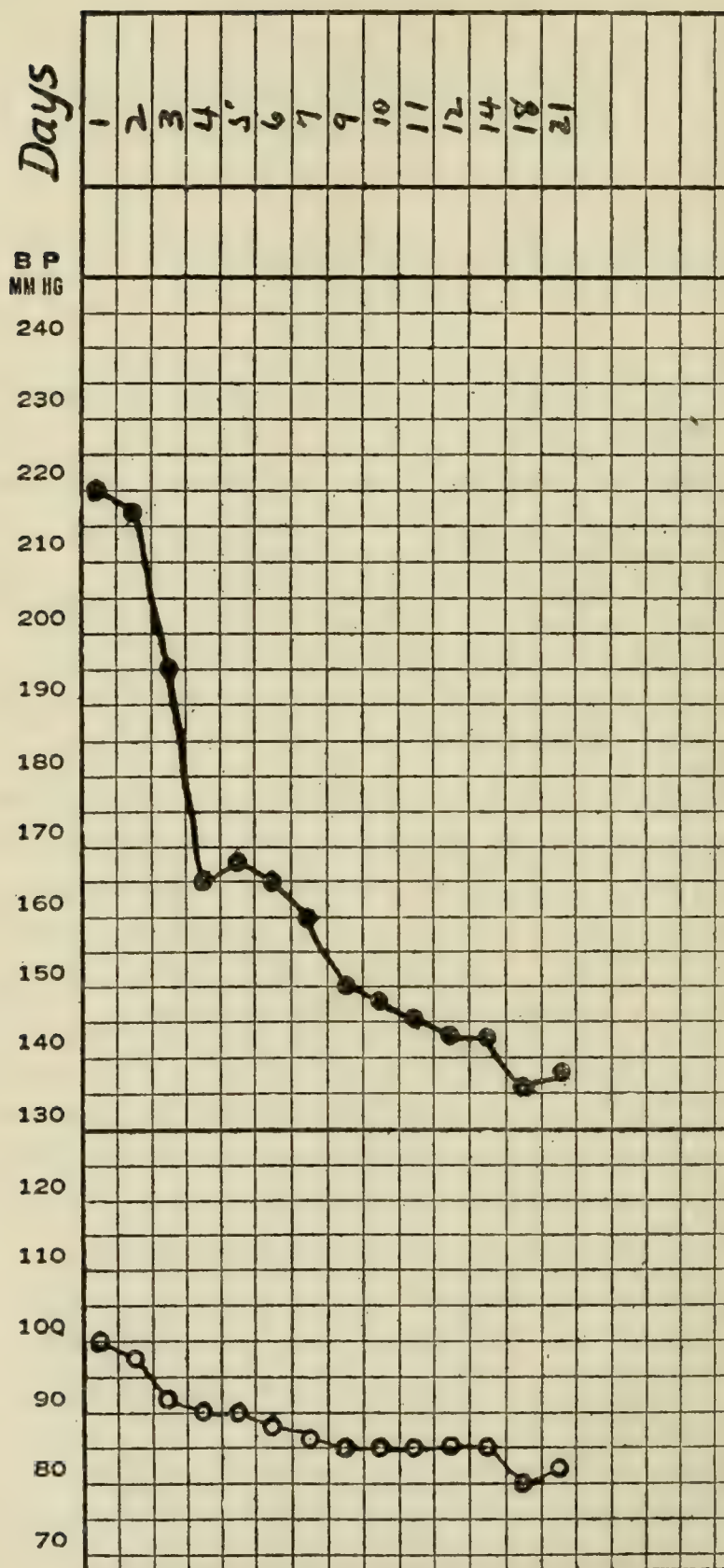
(2) Arterial hypertension does not exert a conservative but rather a destructive influence over the cardio-vascular apparatus.

(3) An effort therefore should always be made to reduce it; if successful the walls of the arterial tree and the cardiac and renal compensation will be conserved and their decompensation relieved or restored. If unsuccessful no harm will have been done to either heart or kidneys or blood-vessels.

(4) Individuals, especially at the close of the third and during the fourth decades of life, and thereafter, should be instructed to report to their physician, as they are told to report to their dentist, in order that arterial hypertension may be detected early, preferably of course, before arteriosclerosis has developed to any appreciable degree; certainly before it has robbed the arterial tree of its elasticity or vasomotor response. For there results then, a set of rigid fixed tubes which can no longer respond to vaso-dilating impulses, causing thereby irreducible arterial hypertension; this in its turn resulting in all the dire aftermath in heart, kidneys and blood-vessels and tending always ultimately toward a fatal termination.

In further conclusion we wish to express our grateful appreciation to our Chief of Service, Dr. George Dock, who has largely made this study possible through placing at our disposal the rich clinical material of the wards and out-patient department of the Internal Medical Service at Barnes Hospital. Also our indebtedness to Dr. H. H. Shackelford for his faithful study of the cases in the out-patient department and his effective work in the preparation of the charts and tables.

CHART VI.



Showing blood-pressure curve in typical case of Group II.

BIBLIOGRAPHY

- ¹ TRAUBE: *Gesamelte Beitrage*, Bd. iii ss 165, 235.
- ² MABOMED: *Guy's Hospital Reports*, 1870.
- ³ RIEGEL: *Berlin Kl. Wochenschr.*, 1882, No. 23.
- ⁴ CLIFFORD ALLBUTT: *Diseases of the Arteries and Angina Pectoris*, vol. i, p. 9, 1915.
- ⁵ LAWRENCE: *Relation of Hypertension to Urinary Secretion*, *Amer. Jour. Med. Sc.*, Sept., 1912, 330.
- ⁶ JANEWAY: *Amer. Jour. Med. Sc.*, 1913, cxiv, 625, 656
- ⁷ MOSCHCOWITZ ELI: *Amer. Jour. Med. Sc.*, clviii.
- ⁸ RINGER, A. I.: *Jour. Am. Med. Sc.*, vol. clxi, no. 6, p. 806.
- ⁹ ALLEN, F. M.: *Jour. A. M. A.*, vol. 74, no. 10, p. 652.
- ¹⁰ HOWELL: *Text-book of Physiology*, 6th ed., p. 838.
- ¹¹ VOLHARD and FAHR: *Die Brightsche Nierenkrankheit*, Berlin, 1904.
- ¹² ADAMI and NICHOLS: *Principles of Pathology*, vol. ii.
- ¹³ CLIFFORD ALLBUTT: *Diseases of the Arteries and Angina Pectoris*, vol. i, p. 192, 1915.
- ¹⁴ HOWELL: *Text-book of Physiology*, 6th. ed., p. 514.

MALARIA RELAPSE DUE TO IMPROPER TREATMENT

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THE case before us will serve as an illustration and basis for discussion of one of the most frequently made errors in the treatment of malaria in this country. What happened in this case is what is going on all over the country where malaria prevails. If the evil were corrected there would result rapid decline in the prevalence of malaria.

The patient, a man about forty years of age, as you see, was admitted to the hospital yesterday afternoon in a very bad condition. His temperature was 106° F., and his condition was such as to lead the admitting officer to suspect that he had meningitis and to send him to the infectious disease ward. Soon after admission, on account of what was considered evidence of meningitis, a lumbar puncture was attempted, but for some reason it was unsuccessful. A blood specimen was taken and upon examination it was found to contain large numbers of tertian malaria parasites.

Just here we may digress to emphasize the large amount of useful information that may be obtained from examination of an ordinary blood-smear. A differential count and examination for malaria on such specimens will frequently give valuable information, as it did in this instance. In fact, in this instance it made a diagnosis at once that could not have been made otherwise. On account of the great value and simplicity of examination of a blood-smear, I believe that in such instances as this it should be done before more formidable procedures like, for instance, lumbar puncture are resorted to.

This patient's temperature returned to normal during the night and is still normal this morning. It seems to be an ordinary case of tertian malaria. The thing that is especially interesting, however, to which I wish to direct your attention, is the history. Upon inquiry, he tells me that he had several attacks of malaria from time to time during last year and that he had one attack lasting a few days, about two months ago. The attack he had two months ago was treated in this hospital. I asked him whether he took quinine when

he was in the hospital and he said he did. I then asked him if he had not taken any quinine since he left the hospital and he said that he did take ten grains daily for two weeks according to the instructions given him by the physician who treated him in the hospital when he was discharged. He says he would have taken it longer if the doctor had advised him to do so, but he is sure that the advice given was to take it for two weeks.

What I wish to bring out is the fact that here is a patient who, after repeated relapses previously, was treated for malaria in this hospital two months ago. He received treatment that was sufficient to relieve his clinical symptoms, but was discharged without adequate treatment to cure the infection. In the natural course of events he has relapsed again and has now returned to the hospital, where he will take up the time of the hospital staff and be considerable expense.

If, on the other hand, he had been given proper advice when he was discharged he probably would not have relapsed and would not have returned for malaria. Not only has he suffered another attack of malaria, with whatever suffering and risk of life may be involved, but wherever he lived he has been a malaria carrier during most of the time and at least a potential source of infection to others. It is quite possible that others have already been infected from him. He had confidence in his hospital physician and would have carried out proper treatment if he had been instructed properly. There is no evidence whatever that he would not have done so; on the contrary, he says that he was willing to do most anything the doctor told him because he was anxious to get rid of his disease.

The standard treatment for malaria recommended by the National Malaria Committee is as follows:

For the acute attack 10 grains of quinine sulphate by mouth three times a day for a period of at least three or four days, to be followed by 10 grains every night before retiring for a period of eight weeks. For infected persons not having acute symptoms at the time only the eight weeks' treatment is required.

The proportionate doses for children are: Under 1 year, one-half grain; 1 year, 1 grain; 2 years, 2 grains; 3 and 4 years, 3 grains; 5, 6 and 7 years, 4 grains; 8, 9, and 10 years, 6 grains; 11, 12, 13, and 14 years, 8 grains; 15 years or older, 10 grains.

Most patients get treatment which relieves the clinical symptoms in comparatively short time and there is at present abundant evidence

that many physicians are adopting the standard treatment. Unfortunately, a great many neglect to advise and impress their patients with the importance of taking quinine for eight weeks to get rid of the infection, as occurred in this case.

After the patient is relieved of the clinical symptoms he is still infected and is likely to relapse sooner or later unless treatment is continued sufficiently long to cure the infection. Some people are cured of their infection in a very much shorter time than others are. If we had any way of knowing the individual who would be cured by a short period of treatment we would of course advise only the necessary period of treatment. Unfortunately, we have no way of determining whether a given individual will be relieved in a short time or will take a much longer time to be cured of his infection. Therefore, the only proper thing to do is to advise all patients to take quinine for a period that is known to be long enough to cure the infection in practically all cases. That period is eight weeks.

I maintain that a physician has not discharged his duty to his patient until he advises him to take quinine in proper doses for eight weeks following relief from clinical symptoms of malaria. To discharge a patient, as was done in this case, with instructions to take quinine for a short period like two weeks, is certainly not giving the patient what he has a right to expect when he employs a physician.

A great many physicians have the idea that patients will not take quinine this long and it is a fact that there are occasionally instances in which they will not. It is usually the physician's fault, however, and not the patient's fault. To prescribe or issue a sufficient amount of quinine for eight weeks and tell the patient he should take it, without giving any further explanation, is not sufficient. If, on the other hand, the physician deliberately explains to the patient so that he understands that it is necessary to take quinine for this length of time for the purpose of getting rid of the infection, after the clinical symptoms have been relieved, he usually will carry it out. If no explanation is given, when the patient gets to feeling well and so far as he knows is perfectly well, he naturally quits taking the quinine. He does not understand the nature of the disease nor the object of the medication following relief of the clinical symptoms.

SUMMARY

We may summarize the facts in this case and the lesson it is intended to bring out about as follows: We have a patient who has ordinary tertian malaria who had a number of relapses last year and now has the second relapse that has occurred during the present year. He was treated in this hospital some two months ago and discharged with instructions to take ten grains of quinine daily for a period of two weeks. He followed instructions faithfully and has now returned to the hospital with another severe attack which is reasonably certain to be a relapse. The patient was not given the benefit of the standard treatment for malaria which he had a right to expect from the physician who treated him.

The standard treatment for malaria is as effective as any other method of treatment known and more effective than most other methods. On account of this fact, a malaria patient who consults a physician is entitled to its advantages. In fact, I believe that the time is not far distant when a physician who does not give his malaria patients the advantages of the standard treatment for malaria will expose himself to just criticism and perhaps even worse in the event there should be unfavorable developments subsequently.

SOME CASES OF THE APOPLECTIC FORM OF TUBERCULOUS MENINGITIS IN ADULTS

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No matter how sudden may have been the onset of the apoplectic phenomena observed in the cases of tuberculous meningitis that shall be related, there is not among them a single instance in which the tuberculous meningitis had not made its presence manifest for a certain time by prodromata which were highly suggestive for the diagnosis. In Case I, the patient had complained of headache for two days, as well as insomnia and general weakness, while the evening preceding the apoplectiform seizure there had been vomiting. In Case II the patient had presented changes in character although there was no apparent disturbance in the general health. In Case III, disturbances of the intelligence had been noted for two months, the patient accusing her parents of wishing to do away with her by taking her life. In Case IV, the patient had become very excited and exalted for several days prior to the seizure and said she saw double. In Case V, there was repeated vomiting before coma took place. Finally, in another patient nervous tremor had been noted, while if in others premonitory symptoms are not noted in the case history it is because there was absence of necessary data at the time the patient was admitted to the hospital.

Therefore, prodromata are rarely wanting in the patient's history prior to the apoplectiform seizure. The classic writers have especially drawn attention to those immediately preceding the attack. On the other hand, Chantemesse has endeavored to demonstrate the importance of prodromata appearing long before the onset of the apoplectic manifestations, such as a change of character which has been noted in several cases before there was any manifestation of the affection either in the brain or lungs. Since all of these patients died sooner or later from tuberculous meningitis, it is rational to suppose that the previously appearing cerebral disturbances were dependent upon a meningeal tuberculous process undergoing its evolution. Consequently, various and very different symptoms such as headache,

vomiting or change of character, should cause the physician to be on his guard when a patient develops a sudden apoplectiform attack, with muscular resolution and intellectual disturbances which go with it and give rise to the clinical picture of an apoplectic seizure.

In Cases II and III, the attack occurred in the night. When the patients retired for the night they appeared to be perfectly well and offered nothing abnormal. In the morning they were found in deep coma. Andral's patient presented all the typical symptoms of an attack of apoplexy; loss of consciousness lasted some twenty hours. Another patient was found in a state of stupor in the morning, with incontinence of urine and weakening of the intellectual faculties. Still another patient sat up in bed in the middle of the night proffering some incoherent words and then suddenly fell back on the bed in a state of coma.

Finally in Case I, the onset of the attack took place in the middle of the night and in the morning the patient was found in the dorsal decubitus, insensible to everything going on around him with eyes wide open and not appearing to hear the questions put to him. Muscular relaxation was general; sensibility persisted but in a lessened degree and the patient reacted when the skin was pinched. Respiration was noisy although not stertorous.

This clinical description, with certain variations, was present in other cases here reported. In one case hydrencephalic cries were noted, or the head and eyes were found in conjugate deviation; occasionally there was rigidity of the neck muscles so that the head could only be placed in its normal position with difficulty. In other cases muscular resolution was more marked on one side of the body and at the same time there were symptoms of facial hemiplegia. Finally, in Case VII, the attack assumed a convulsive character—there was a true epileptiform seizure which was followed by a left-sided hemiplegia.

Later on in the evolution of the process the clinical picture will vary somewhat according to the rapidity of the evolution of the affection and from this viewpoint these cases may be classified under three headings. In the first, muscular resolution remains complete, stupor persists and becomes more profound, the respiration becomes stertorous, there is incontinence of both bladder and rectum, death

taking place in coma in twenty-four to forty-eight hours (Cases I, V, VI).

In the second group of cases, the patient appears to come out of his torpor but remains somnolent. One side of the body in hemiplegia and contractures are occasionally noted. This hemiplegia rapidly decreases and may even disappear but during this time the general condition deteriorates. The patient develops loquacious or very restless delirium in the evening and keeps up a ceaseless muttering of words. Respiration becomes accelerated and labored, death taking place in from four to eight days.

Sometimes life may be prolonged after the apoplectic attack. Andral's case is an example. The apoplectic seizure resulted in paralysis of the right side of the body but disappeared in a few days; the intelligence remained obtuse, there was mild delirium, the temperature went up and finally about a month later another attack of apoplexy occurred which resulted in death.

Death is of course, the ultimate outcome, but may not occur for twenty-four hours, a week and exceptionally a month after the apoplectic seizure.

I shall now refer to the indications furnished by the temperature, pulse and respiration during the evolution of the apoplectic accidents. The temperature was studied in three cases (Cases I, II and IV). In Case I, it was 39.3°C . (101.5°F .) on the morning of the attack and 41.2°C . (106.2°F .) in the evening, a few hours before death occurred. The tuberculosis had been pyretic during its entire evolution, consequently the temperature of 101.5°F . was in no way characteristic.

In Case II, the temperature was 37°C . (98.6°F .) for two days before death while on the day the patient died it was 40.4°C . (104.6°F .). In Case IV, the temperature registered 38.4°C . (101°F .) two days before death and 40°C . (104°F .) on the day it took place. Only one conclusion can be made, namely, that the temperature rises as death approaches.

The pulse offers variable characters, usually being increased in frequency, sometimes irregular, small or even imperceptible. The respiration presents one constant phenomenon, namely frequency, which is usually at least forty respirations or more to the minute and Cheyne-Stokes rhythm is sometimes met with. Briefly, respira-

tion is usually accelerated, the temperature rises toward the end and the pulse offers great differences in type, varying in character during the evolution of the process.

The type of tuberculous meningitis under consideration may be primary or secondary. In the latter case, no matter what anomalies exist at the onset or during the evolution, the diagnosis will be made especially by auscultation and a careful general examination of the patient for Pott's disease, coxalgia, adenitis, etc. A search for the tubercle bacillus in the sputum and an examination of the fundus oculi should always be made and will often give very valuable diagnostic data.

Tuberculization of the meningitis should always be suspected when, in a phthisical subject, cerebral accidents supervene unexpectedly. However, it should not be forgotten that in cachectic tuberculous patients thromboses and emboli may develop which have nothing to do with tuberculous meningitis, and in this respect Hayem has recorded a fine example. A tuberculous patient had seven or eight dizzy spells and died suddenly in the last one. At autopsy a thrombus in the right brachio-cephalic trunk was found which had been the origin of multiple small emboli.

After a certain age, the coexistence of cerebral softening with phthisis is not impossible, the following instance reported by Chouppe, being a case in point. The patient had phthisis, and in his antecedents there was a history of sudden loss of consciousness which lasted for some time although the patient could give no exact data relating to it. He recovered and the morbid incident left no traces. Eighteen months later the patient died from his pulmonary tuberculosis and at autopsy no trace of meningitis could be detected but there was a superficial cerebral softening involving the right sphenoidal lobe and the left frontal lobe at the level of the third convolution. The lesion was an old one, grayish in color. Microscopically the lesion was a pure softening of the brain.

But it is the primary forms of tuberculous meningitis in the adult that especially give rise to much difficulty in the diagnosis particularly when the process assumes an irregular evolution. It often happens that the clinician maintains a prudent reserve and suspects the diagnosis rather than affirming it. I will briefly consider some of the affections which can be mistaken for the apoplectic type of tuberculous

meningitis and will compare them with this process as it occurs in adults, whose onset is sudden and unusual and from this comparison I shall endeavor to extract some elements for a differential diagnosis.

Cerebral growths present a number of symptoms which are common to tuberculous meningitis—headache, vomiting, epileptiform convulsions, paralyses—and when during their evolution an apoplectic seizure arises, the difficulty may be great. But the apoplectiform paroxysms observed in cerebral neoplasms offer a short period of coma; there is rather a progressive weakening in a limb than true paralysis. The patient rarely dies in the first attack and those that follow usually increase in intensity. Then too, in some cases the patients' antecedents will point to the diagnosis. He may have had syphilis, in which case an exostosis or gumma will naturally be thought of or a carcinoma operated on previously will lead to a probable diagnosis of secondary malignant cerebral growths.

Brain abscess may also present an apoplectic phase, but there will be a history of fracture of the skull or otitis media, a more special localization of the pain and a low but progressive rise of temperature.

Acute purulent meningitis gives rise to headache, vomiting, delirium and dissociation of the pulse as in tuberculous meningitis. It has been known to offer an apoplectic onset. But the cephalalgia is very severe in acute meningitis and the temperature at the very onset of the process reaches about 40°C. (104°F.).

Cerebral hemorrhage does not usually cause headache or vomiting; the apoplectic seizure is followed immediately by a drop of two or more degrees of temperature below normal, while the consecutive paralysis is slower to retrogress than in tuberculous meningitis.

Cerebral softening is preceded by prodromes, such as headache, and vertigo. Vomiting does not occur and the apoplectic seizure is incomplete. When the softening is the result of a thrombosis, the onset is slow, progressive, the seizure slowly becoming complete and sometimes the patient feels it coming on. He is usually advanced in age with signs of arteriosclerosis.

When the softening is the result of an embolus the heart will usually present lesions and generally the paralysis is right-sided with aphasia. Signs of visceral infarctus are likewise present.

Meningeal hemorrhage arising during the evolution of pachymeningitis can only be suspected during life on account of its extreme

rarity. The seizure offers the same symptoms as tuberculous meningitis. At the most the distinguishing signs will be a weakening of the intelligence and a history of alcoholism, the most frequent cerebral condition met with in pachymeningitis.

Tertiary cerebral syphilis may give rise to apoplectic symptoms when, for example, a gumma of a vessel is the starting-point of a thrombosis. The pathogenesis of the accidents can only be obtained from the patient's history, or if data are wanting the Wassermann will help should it prove to be positive. In syphilis, a gradual decrease of the sight will be noted and the paralysis of one of the eyelids, or rather an impossibility to move it, than a true paralysis (Mercier).

Apoplectiform cerebral congestion described by Trousseau may occur in acute alcoholism or following insolation. The characteristic odor of the breath in the former and the knowledge of the cause in the latter, will lead to the diagnosis. For that matter, coma is less deep in cases of congestion and the hemiplegia resulting is quite temporary.

The comatous accidents may be attributed to uremia, especially if there be morbid changes in the kidney known to exist. But the coma of uremia does not give rise to true paralyses or contractures while the temperature is invariably very low. When the case is one of diabetic coma, the condition will have been preceded by gastrointestinal disturbances and especially dyspnoëic accidents, convulsions and contractures are always wanting, while the examination of the urine is characteristic.

Finally, *general paresis* and *sclerosis en plaques* may give rise to apoplectic seizures during their evolution which might be mistaken for the apoplectic form of tuberculous meningitis, but in the two former morbid processes the duration of the seizure is short while examination of the patient will easily reveal the causal affection.

I need hardly insist upon the special gravity that an apoplectiform attack gives to the prognosis of tuberculous meningitis, and in two of the causes to be given, death ensued twenty-four hours after the seizure had taken place. In Case II, death occurred three days after the apoplectiform attack; the meningitis was primary. On the other hand, in Gombault's case the meningitis was secondary to a Pott's disease and death took place nine days after the attack.

As particularly serious symptoms I would mention a considerable

rise of temperature and stertor, whose apparition is invariably soon followed by death.

There is nothing special to say as to the treatment excepting that when the slightest doubt exists as to the nature of the process or its possible syphilitic nature, an energetic antisyphilitic treatment should be given without loss of time.

CASE I.—Male, *æt.* 25 years, admitted to hospital March 9. The family antecedents of the patient are replete with tuberculosis. The patient's father was tuberculous and died from hemoptysis; an aunt lost four children from meningitis.

The patient takes cold easily in winter, has coughed for a year, but it has been especially during the past four months that the frequency in coughing has increased and the general state of health has been declining. There was an evening rise of temperature, profuse nocturnal sweating, quite marked emaciation and loss of strength. Has never had hemoptysis.

Status praesens.—Voice hoarse, and weak. The larynx has been involved for about three months. Temperature: Morning 38.4°C. (100.8°F.); evening, 39.2°C. (102.8°F.). Profuse expectoration of nummular sputum.

Examination of thorax.—The ribs and clavicle project. Incomplete percussion dullness in the left supra-spinous fossa. Inspiration and expiration notably weak; the respiration is whistling and subcrepitant râles can be heard when the patient coughs. There was the same incomplete dullness in front with many subcrepitant râles in the apices.

There was incomplete dullness over the right infra-spinous fossa, with decrease of sonority down to the spine of the scapula. Respiration short and jerky, while over the supra-spinous fossa there was incomplete dullness and a weak respiration. When the patient coughed, large moist subcrepitant râles could be heard and after coughing the respiration became blowing. There was supplementary respiration at the right base.

In front over the clavicular region there was incomplete dullness, diminished inspiration and prolonged expiration. A few subcrepitant râles when the patient coughed. The sputum contained quite a large number of tubercle bacilli.

Treatment.—Calcium phosphate, 4 grams; cod-liver oil and creosote.

March 15.—Patient in good condition, coughs less. Allowed to get up during the day. But the temperature remained high, around 39°C. (102°F.) or more in the evening, 38°C. (100.8°F.) or more in the morning. Antipyrine two grams.

The patient's condition remained about the same although he seemed to gain strength, but on March 25 he complained of headache for the first time which had prevented him from sleeping. The pain was not severe. Evening temperature 39°C. (102°F.), morning 38°C. (100.8°F.).

March 26.—The headache continued although not more severe. The patient, who was intelligent, complained of prostration and loss of appetite. In the evening there was some vomiting, at first mucous and then bilious. Evening temperature 39.3°C. (103°F.), morning, 38.4°C. (101°F.). Pulse 96, regular. No diarrhœa.

March 27.—In the morning patient found in complete stupor. Temperature 39.3°C. (103°F.), pulse 112. The patient was lying on the back, eyes open and motionless. No unequalness of the pupils. Does not respond to questions. Sensibility persists; withdraws limbs when pinched and shows evidence of suffering.

Neither the limbs nor neck are stiff. No deviation of the head or eyes. There are occasional jerky movements in the limbs, more particularly of the arms which present slight contractions. These are the same on both sides; no hemiplegia.

The patient had slight epileptiform paroxysms during the entire morning. At noon there was complete coma. Respiration became stertorous; incontinence of urine. Temperature 41.2°C. (106°F.) at 5 P.M.; pulse frequent and very small. Died without change at 10 P.M.

Autopsy thirty-six hours after death. *Lungs.*—The left upper lobe contained several cavities the size of a walnut, the surrounding parenchyma was infiltrated with granulations. In the right apex were some caseous cuneiform tuberculous masses with their base directed toward the pleural surface, with disseminated granulations around them. Simple congestion in the lower half of this lung.

Liver weighed 1920 grams, brown in color; does not look like a fatty liver.

Kidneys hyperemic; no tubercles. Left kidney weighs 215 grams, right 190 grams.

Heart very slightly enlarged. No valvular lesions, no fatty infiltration, no atheroma of aorta.

Brain.—At the base of the right hemisphere at the level of the occipito-temporal convolutions exists a large superficial focus of softening, 10 centimeters long and about 5 centimeters wide; the arachnoid is adherent over this focus.

On section the area of softening did not extend beyond the gray matter; it formed a reddish gelatinous mass, dotted in parts with darker spots which were almost black. However, in the anterior and external portion of the focus at the level of the sphenoidal horn, the softening involved the subjacent white matter. At the borders of the focus the pink tint faded away and the surrounding brain tissue was normal in consistency.

At a corresponding point of the left hemisphere the meninges were slightly adherent to the subjacent parts to the extent of a twenty-five cent piece; the superficial part of the convolutions was alone the seat of commencing softening.

There was little fluid in the lateral ventricles. At the upper part of the right ventricle were some small superficial erosions.

In the large focus in the right hemisphere the meningeal vessels, especially the veins, were gorged with dark blood, while along their courses gray graulations could be seen contrasting distinctly against the vascular arborizations. The surface on the left lobe offered the same venous turgescence. Over both lobes the meninges were very thick and offered areas of slightly opaline hue but these did not adhere to the subjacent structures. When examined by transmitted light they presented several areas of gray granulations along the vessels. There was nothing special over the Sylvian fissure or the fronto-parietal convolutions.

The branches of the posterior cerebral artery were examined *in situ* to ascertain if there was not an occlusion due to compression of the tuberculous nodules, but the result was negative. Macroscopically the lesion was a focus of acute red softening.

Microscopically the classic lesions of tuberculous meningitis were found. The pia was infiltrated with embryonal cells. There were numerous young tubercles around the vessels, their internal tunic

was thickened, in other words a tuberculous periarteritis and endarteritis. The lymphatics were filled with embryonal cells.

Sections made perpendicularly to the surface of the brain in places corresponding to the punctuated hemorrhagic areas seen in the gray matter, revealed numerous capillary hemorrhages. At some spots the artery giving rise to the hemorrhage could be seen in the center of the foci with its lymphatic sheath gorged with red blood corpuscles. The red blood corpuscles were contained in meshes of fibrin, the latter being distinctly granular in places. Somewhat more refringent spots which seemed to be granular bodies were disseminated irregularly in some of the sections.

CASE II.—Female, *æt.* 37 years, entered hospital July 22. The data obtained from the patient's sister were very vague. She had been ill for two years and certain changes in her character had been noted, although she continued her work. For the past year the menses have been suppressed. On July 21, she was found unconscious and on the next day she was admitted to the hospital in complete coma. The patient was quite stout and of a robust constitution.

July 22.—Evening temperature 37°C . (98.6°F .), pulse 108, regular respiration, 39. Coma without stertor. Head rotated to the left, slightly inclined on left shoulder; it can be brought to the median line with difficulty and as soon as released resumes its former position. The eyelids move, usually remaining half opened; both eyes are deviated to the left. The right naso-labial furrow appears to be less marked than the right; the nose is slightly drawn to the left. The lips are partially opened, but the lower lip does not drop. The lower jaw can be lowered with difficulty. Swallowing was difficult.

The left arm and leg execute a few spontaneous movements and do not fall when raised. The movements also exist, but to a less extent, in the right limbs. Tickling the plantar surface of the feet provokes reflex movement of the lower limb. Pinching the legs also causes reflex movements and complaints from the patient. The right lower limb is extended and slightly contracted, the right upper limb is likewise contracted, the forearm is bent at a right angle on the arm, the four last fingers are flexed on the palm, the thumb extended on the index finger. There is some resistance when an attempt is made to extend or bend the forearm. When the limb is raised and left alone, it remains raised for a few instants, and

while in this position is the seat of slight tremor. The upper left limb is not contracted to the same extent and movements of extension and flexion are quite easy. Nothing in the heart; abdomen distended; a small violet-colored spot over the sacrum.

July 23.—Respiration 37 to 39. Pulse 102, unequal. The head is *not* deviated to-day. Eyes half opened; nystagmus. Sensibility to pain preserved on left, decreased, *but not abolished*, on right, when the right arm is raised it falls inert. There are some spontaneous movements in the left limbs. Slight cyanosis of face; some oedema of the malleoli.

One hour after the above notes were taken the right upper limb when raised fell slowly after remaining raised for an instant, as on the day previously, while one hour later it was again placid. The condition was the same in the evening. Pulse 120, very irregular. Very marked right facial paralysis.

July 24.—Respiration, 48; temperature, 40.4°C. (104.8°F.); pulse 108, very small, irregular, hardly perceptible; coma. The breathing was louder than yesterday, although not stertorous; the patient sighs occasionally. The right upper limb can be raised without resistance and falls in a state of placidity. The left upper limb offers some resistance when raised and falls less rapidly.

The reflex movements produced by tickling the plantar surface of the feet are less pronounced on the right than on the left. The right pupil is more dilated than the left; the face is cold, the lips cyanosed; neck stiff; abdomen distended. The erythematous patch over the sacrum has sloughed in the center. The patient died at 1.30 P.M. following convulsive seizures of a few minutes duration.

Autopsy, July 2.—*Skull.* Small ecchymosis under the pericranium over the right frontal bone. On its internal aspect the frontal presents whitish areas with irregular borders, and over these areas on the external aspect of the bone the latter is more vascularized. The cerebral veins are filled with fluid blood; the upper longitudinal sinus contains a recent clot. The cerebrospinal fluid is cloudy.

The meninges of the under surface of the brain are thickened, opaque, especially in the median line and contain gray tuberculous granulations which are appreciable only on the layer covering the anterior subarachnoid space. The meninges of the under surface of the bulb, protuberance, especially on the sides, are very thick. The

pia at the bottom of the Sylvian fissures, especially the left, is also very thick. At the origin of the left fissure there is a grayish-yellow patch which appears infiltrated with pus.

On the under surface of the frontal lobes especially the left, are purulent fibrinous exudates the size and shape of millet seeds with irregular contours and connected with each other by small whitish-yellow spots of the same nature. There is also a purulent thickening of the meninges on the under aspect of the sphenoidal lobe.

On other parts of the brain, the meninges are hyperemic and offer some rare fibrinous deposits along the course of the vessels. Nothing to note on sections of the brain.

The internal surface of the dura of the cord is covered with very fine tuberculous granulations. There are likewise numerous granulations on the posterior aspect of the cord.

Hypostatic congestion of the lungs which are free from tubercles. The other viscera are normal.

CASE III.—Female, *æt.* 32 years, entered hospital on April 1. The patient's mother stated that about two months previously she noted that her daughter's mind was affected although her general health did not seem changed. The patient accused her family of wishing to do away with her. Patient had always appeared to enjoy good health and never indulged in any form of alcohol.

On March 29, she was found at the foot of her bed unconscious; she did not open her eyes for about two hours. Then she regained consciousness and spoke some words which could not be understood by those about her. For three days she remained in the same condition although no paralysis was noted. The hands, especially the left, were animated with little movements of prehension. There seemed to be cutaneous hyperesthesia. No stool was passed and she did not vomit. As her condition remained unchanged she was brought to the hospital.

When admitted the patient was in a stupor, the eyes open, pupils quite immobile and rather contracted; light did not seem to affect her painfully. The head was strongly inclined to the right shoulder and when an attempt was made to straighten it pain was provoked, causing the patient to cry out. It was difficult to place the patient in a sitting position. The cervical spine was very stiff. There had been no vomiting and the patient could swallow well. No paralysis of the limbs and when pinched each limb was quickly and forcibly with-

drawn. The sensibility appeared to be quite exaggerated, especially low down on the thighs, where the slightest pressure caused the patient to make a grimace.

There was no cough and auscultation revealed nothing abnormal in the lungs. The abdomen was soft and otherwise normal.

April 2.—No vomiting; speech has somewhat returned and the patient seems to understand. A happy delirium has developed and the other symptoms persist with the same intensity. Death took place at 11 P.M.

Autopsy thirty-six hours after death. The meninges are thick and infiltrated. Here and there over the convexity and especially in the fissures numerous granulations of extreme tenuity were found on the vessels; only two were of some size and these were on the under surface of the right posterior lobe. The ventricles were dilated and filled with clear fluid, while on the choroid membrane were numerous extremely fine granulations. The fornix of the brain was very softened; at some spots the peripheral gray matter was softer than normal. The cord offered several distinct granulations.

Both lungs were riddled with miliary granulations. Heart large. Granulations scattered over the surface of the liver and spleen. No trace of tubercles was found elsewhere.

CASE IV (notes communicated by Dr. Rombault).—Female, *æt.* 14 years, transferred to medical section October 23. Patient's father died insane, mother in excellent health.

The patient had been in the surgical section of the hospital since September 23, under treatment for a cold abscess of the neck due to *spinal caries*. During her entire stay there had been no symptoms in the nervous system, and it was also known that she had never presented convulsive seizures or coma.

For the past few days the patient had been somewhat exalted and said she saw double, when in the night of October 23 at about 2 A.M. she suddenly sat up in bed, proffered a few incoherent words and then fell back in a coma. The interne on duty made the following notes at the time: Absolute loss of consciousness, stertor and foam at the mouth. Relaxation complete on the entire left side and facial paralysis on the same side, rotation of the head and eyes to the right.

The patient made some automatic movements, passing the hand

over the right side of the face. The left eye was wide open while the right upper lid drooped. The pupils were widely dilated, equal and did not react to light.

The following morning the patient had recovered consciousness but she was still delirious and stupid. The left hemiplegia persisted without contracture. The left lung only was filled with large noisy râles. It was then that the patient was transferred to a medical ward.

In the evening there was no trace of coma, but the patient was stupid, replied to questions badly and appeared to have no idea as to what had taken place. Flaccid paralysis on left side; face markedly deviated to the right. Temperature about normal.

October 24.—Movement has returned to a slight extent on left side.

October 25.—Facial paralysis still persists, but the left arm and leg have regained their movements. Stupor less pronounced. The patient remembers that she was in another ward.

October 26.—Facial paralysis much diminished. The uvula is deviated to the right but the velum is not deformed. Speech distinct.

October 28.—Patient was restless all night; tried to get out of bed. In the morning the skin was hot, the gaze somewhat bewildered and she replied badly to questions. In the evening, she sat up in bed the face animated and incessantly muttered words without meaning. Swallowing disturbed; the cough is typical of paralysis of the larynx. Speech is often cut short by frequent inspirations. Patient finds the name of objects shown her with difficulty. Axillary temperature, 38.5°C. (101.4°F.).

October 29.—Somewhat quieter, somnolent. Skin hot. Temperature, 37.5°C. (99.4°F.); pulse 132, intermittent. Cyanosis of face. Paroxysmal cough, respiration free, some loud breathing in right apex. Evening temperature, 38.4°C. (101.2°F.); respiration 44; pulse so small that it cannot be counted. Somnolence more pronounced. Cold sweat on face which is violet color. Noisy respiration.

October 30.—General cyanosis. Very rapid respiration. Voice extinguished. Somnolence. Evening: Pulse 120; respirations 60. Extremities cold.

October 31.—Cyanosis still more complete. Delirium less. Evening: Somnolence, automatic movements, incoherent words.

Temperature, 40°C. (104°F.), respiration 48, pulse cannot be counted. Death at 9.30 P.M.

Autopsy on November 2.—*Lungs*.—Quite extensive bronchopneumonia, but no granulations. Some caseous foci in neck due to tuberculosis of dorsal vertebræ. At this level there was a pachymeningitis but the cord was normal.

Brain.—The entire region of the hexagon was occupied by a very dense greenish exudate in which the vessels and nerves were embedded. Behind, it extended on to the protuberance and flattened out the basilar artery in which a small clot was found. Then it extended on to the bulb and here the nerves were embedded in a pinkish colored exudate. The remainder of the pia was very vascular in relation to the more or less abundant exudate according to the regions.

In the Sylvian fissures and along the olfactive bulbs were numerous granulations, usually small, lying along the vessels. The ventricles were not distended. There were no morbid changes in the brain other than a somewhat rosy tint of the cortex.

CASE V.—Male, *æt.* 17 years, entered hospital January 7. The patient was pale and thin and presented a swelling of the right knee which prevented him from walking. There was also a tumefaction over the internal condyle of the tibia.

For several years the patient had had hemoptyses, nocturnal sweats and diarrhœa, while emaciation was progressive. On auscultation a slight souffle could be heard in both apices with some large mucous râles throughout the rest of the lungs.

Tumefaction of the abdominal, inguinal and iliac lymph-nodes. There had never been any cerebral symptoms in the patient's antecedents.

Treatment.—Cod-liver oil, rest in bed, tonics. The patient improved somewhat, but on February 4, he was seized in the afternoon with repeated vomiting following which complete coma developed and from which he could not be aroused.

The patient remained on his back with the head thrown backward, the eyes turned upward and gave vent from time to time to the meningeal cries met with in tuberculous meningitis in children.

February 5.—The stools were passed involuntarily and the patient died on the following day at 1 A.M. in deep coma.

Autopsy thirty-two hours after death. The dura offered nothing

in particular, the arachnoid was thick. The subarachnoid fluid was rather abundant, both in the sulci of the convolutions and ventricles. The pia was infected and the seat of tuberculous granulations. These granulations were scattered over nearly all the pia although they were in greater number in the Sylvian fissures where they were remarkably confluent. They were dirty white in color and contrasted well against the infected membrane. The brain was finely infected in the cortex which everywhere presented a grayish color tinted red.

Granulations and caseous foci in the lungs. Caseous mesenteric lymph-nodes, intestinal ulcerations.

Remarks.—Besides the anatomical lesions, there is a clinical feature in this case which I think should be noted. It is the abnormal form, if I may use this expression, of the meningitis which killed the patient. In fact he died in less than forty-eight hours after the onset of the accident. On the other hand, this evolution, which was rather overwhelming to say the least, belongs to childhood and in young subjects death ordinarily occurs in the midst of convulsive seizures. Finally, although an abnormal amount of fluid was present in the ventricles, it has been shown long since by Piet that this lesion only exists in 50 per cent. of the cases and is itself insufficient to explain death from cerebral compression in only about one case in eight. In the case under consideration the very advanced state of cachexia was probably the real cause of death.

CASE VI.—Male, *æt.* 40 years, presented evidences of advanced pulmonary tuberculosis, entered hospital February 17. Since January 7, last, the patient had presented symptoms of a right-sided sciatica; the pain was intense with only temporary remissions and this only by the use of opiates.

The patient was put upon the usual treatment for sciatica and a notable improvement was obtained which continued until April 14, on which day the patient was seized with an attack of suffocation, with general muscular resolution, stertorous breathing and froth at the mouth. Examination showed that the respiration was the Cheyne-Stokes type, coma was profound and the patient died the same day without regaining consciousness.

Autopsy done on April 16, revealed, besides the pulmonary tuberculous lesions, a tuberculous meningitis with the superficial disseminated foci, without any notable adhesions. The tubercles

were especially marked over the base, but they were not in large number anywhere. The ventricles were greatly distended with fluid.

CASE VII.—Male, *æt.* 33 years, entered hospital on March 23. The patient was a very well-developed subject with a projecting chest. He had never been ill, but for some time past he had indulged excessively in coitus and alcohol; he has had a tremor for some time. He states that he has had two or three epileptic seizures, one three years previously which was very severe, and a milder one three months ago.

On March 17 the patient was seized with an epileptiform attack with loss of consciousness, which lasted nearly an hour and it was then found that the entire left side was paralyzed. The patient was profoundly prostrated, indifferent to everything and vomited several times during the night. A physician summoned found a paralysis of the left side, stertorous breathing and the eyes closed. The patient was freely bled at this time.

When he entered hospital on March 23, the patient was in the following condition. He lay on his back, the head thrown slightly backward; stertorous breathing and complete left-sided hemiplegia. He was unconscious of everything about him and only gave evidence of pain when the skin was pinched. The eyes were closed and bleared and when forcibly opened there was a confused stare. Pupils dilated. Difficulty in swallowing.

The patient was again bled and calomel ordered. This condition continued during the following days. There were some convulsive movements on the non-paralyzed side. Carphology. Death on March 26, at 11 A.M.

Autopsy.—General purulent meningitis with false membranes and adhesions between the cerebral convolutions. Adhesions between the pia and brain. In the anfractuositities and on the surface of the convolutions many tubercles were seen, and a large number were found in the Sylvian fissures. The internal surface of the ventricles rough and granular and they were filled with a large amount of cloudy fluid.

On the right, outside of the optic layer, was a blood-clot the size of a filbert in the midst of a small focus of softening. The white matter was spotted with fine red points. Quite numerous miliary tubercles in the apices of both lungs, while two or three small ones were found in the kidneys. The liver was yellow, hypertrophied and fatty.

CASE VIII.—Male, *æt.* 26 years, entered hospital on March 2. Both parents died from phthisis. The patient presented pulmonary tuberculosis in the second degree. The affection began fifteen months previously. For the past two months he has also experienced pain on micturition and he has also passed blood from the urethra on several occasions. The urine contains albumin.

March 11.—The patient was found in a state of complete stupor with weakness of the limbs on the left side, incontinence of urine and decrease of the intellectual faculties. These symptoms increased during the three ensuing days. The right-sided hemiplegia became more and more marked, while the left limbs also became paretic. There was a complete paralysis of the right side of the face and deviation of the labial commissure to the left. Convergent strabismus of the left eye. Both pupils dilated, especially the right one.

The prostration progressed, the patient no longer replied to questions; he muttered continually. Sensibility was less, but nevertheless was fairly well preserved. No convulsions or vomiting; some diarrhœa. This state of affairs lasted three days. On March 14, at 5 A.M. there was an abundant hematuria and another at 9 A.M. Coma and delirium. Death at 11 A.M. It is to be noted that throughout the pulse was regular and varied between 72 to 84 pulsations per minute.

Autopsy.—The meninges were much infected, especially over the Sylvian fissures, and here they were thickened and so adherent that the fissures could only be exposed by the knife. On the convex surface and surface of the left frontal lobe over the first convolution, the meninges were very thick with milky patches towards the center. At this point there were a large number of submeningeal tubercles separated by numerous vessels along which much pus was seen. The meninges were very congested at this area.

Sections of the brain made at this area revealed a focus of capillary apoplexy under the patches, surrounded by distinct hemorrhagic dots. All this portion of the brain had undergone softening. The focus corresponded to the most thickened part of the meninges and in depth was two centimeters and two and one-half centimeters in diameter. Softening of the formix of the brain. Lateral ventricles distended with fluid.

Lungs infiltrated with tubercles and small cavities in the apices. Tuberculosis of kidneys, ureters and bladder.

CASE IX.—Male, *æt.* 33 years, developed all the symptoms of an apoplectic seizure five days before being admitted to hospital. Loss of consciousness lasted about twenty hours. After this he was paralyzed on the right side, then delirium set in and the patient was brought to the hospital, at which time the following notes were made, on February 22.

Face pale; delirium; limbs on both sides moved equally well. Pulse, 72; tongue good. Temperature normal.

February 23.—Eighth day of the illness, delirium persisted. Pulse hard but not rapid. Face expressionless.

February 24.—Intelligence almost restored, replies slowly but distinctly to questions.

February 27.—Delirious again and for the first time the pulse was rapid.

March 1.—Intelligence perfect, pulse rapid, temperature normal.

March 2 and 3.—Completely delirious. Temperature 39°C. (102.4°F.).

March 15.—There has been no change. Intelligence somewhat cloudy; stupor. The pulse varied from day to day but averaged about 75 beats to the minute.

March 16.—The patient suddenly developed deep coma and appeared like a man with a severe apoplectic seizure. He died a few hours later.

Autopsy.—The meninges covering the convexity of the brain are milky white, opaque and thickened and could be removed in one piece from the brain. The under surface of the left hemisphere in its middle portion was covered by the same milky white meninges but the opaque portions were especially evident in the interval of three or four convolutions. The sulcus separating them had disappeared and the convolutions were adherent to each other. In the midst of the infiltrated thickened pia which united them were a number of miliary tubercles, while in the midst of the gray matter of the adherent convolution about a dozen tubercles were seen. The cerebral tissue surrounding them was very vascular and softened.

The lungs presented miliary tuberculosis but no cavities. A suppurating lymph-node was found and was related to an ulcer present in the greater curvature. Tuberculosis of the intestine, mesenteric lymph-nodes, the liver, kidneys and spleen.

CONCLUSIONS

From the foregoing cases I believe we may conclude that tuberculous meningitis in adults may, in some cases, assume a rather peculiar symptomatic aspect which deserves the name of the apoplectic form of tuberculous meningitis. This form of the process is more prone to be secondary but it can be primary and in the latter circumstance the diagnosis offers much difficulty.

Autopsy of these cases shows the presence of brain lesions as well as meningeal morbid changes. The former are usually inflammatory in character, but they may be likewise necrobiotic. Rapid destruction by necrobiosis or encephalitis partially explains the sudden onset of the accidents which characterize the apoplectic form of tuberculosis of the meninges.

TRAUMATIC EPILEPSY

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EPILEPSY due to traumatism is relatively rare, although injuries to the skull, spinal column and other portions of the body are of frequent occurrence. The opinion is current among the laity, and to a slight extent among some of the members of the medical profession, that epilepsy is often the result of traumatism. In securing the history of the epileptic patient, we are struck with the great importance that is attached, by the patient or his relatives, to the alleged casual relationship which they attempt to establish between some injury and the development of this neurosis. Practically every individual has at some time during his life sustained an injury identical in character with the one cited by the epileptic or his friends as the cause of his disease. It is no doubt in the hope of escaping from the supposed stigma of degeneracy or defect that in the minds of the laity has attached itself to the disease, that traumatism is so often assigned as a causative factor.

Research into relationship of trauma to epilepsy has established the fact, as said above, that traumatic epilepsy is a rare disease. When all the facts of head, spinal and other bodily injuries are collected, sifted and weighed, out of the collated medical data of the recent world war, a new light may be shed upon the traumatic types of epilepsy. The findings so far bid fair, however, to sustain the conclusions that have been drawn from former wars and from the observations that may have been made in hospitals for the insane, colonies for the epileptic, and experiences in the industrial world and in the usual practice of medicine. The United States Public Health Service hospitals and other institutions engaged in caring for the sick and wounded veterans of the world war, treat many epileptic patients who are attributing their neuroses to trauma alone, sustained during military service.

This duty presents a problem calling for careful differentiation between traumatic epilepsy and hysteria, hystero-epilepsy and allied

pyschopathic states. The matter of diagnosis will enter very largely into the question of payment of compensation to ex-soldiers. The status of traumatic epilepsy, therefore, aside from its medical aspects, has a new and vital sociological interest, and will have for several years to come.

The following is a very brief review of the most important data that have been collected by various authorities, and these point to the significant fact of the relative infrequency of true traumatic epilepsy.

Gowers in a study of 3000 cases of epilepsy, ascribed 108 cases, or 3 3-5 per cent., to falls or blows on the head.

Wildermuth states that epilepsy is caused by traumatism in 3.8 per cent. of cases. This percentage is based on a study of 189 cases.

Heeres gives a percentage of 4.2.

English states that a study of 300 cases of head injury revealed the fact that twenty-one patients so injured developed epilepsy within a year. Of these, 7 cases were of the Jacksonian type.

The studies of head injuries received in the Franco-Prussian war show that out of 8985 injuries to the head there developed but 46 cases of epilepsy.

Spratling has observed, in a study of 814 cases of epilepsy in the male, that 8 1-2 per cent. were due to traumatism, and in a similar study of 509 female epileptics he found 3 1-2 per cent. were due to injury.

He also made the observation that male epileptics offered a greater chance for surgical treatment than did cases among women. He makes a qualifying statement, however, in discussing traumatism as an etiological factor, which leads the reader to believe that the relatively high percentages of traumatic epilepsy are somewhat questionable. He studied a highly selected group in institutions, and this no doubt explains the fact that his percentages are slightly higher than those of other authorities. He states: "It cannot be said that trauma was the sole cause of epilepsy in all cases, but it was unquestionably a contributing factor, when it did not act alone."

It will be well, for the sake of accuracy, to establish just what is meant by traumatic epilepsy. Certain definite factors are to be conclusively proven before we are justified in classifying any case within this category. The following postulate is warranted:

First.—The fact must be proven that the person never had, pre-

vious to the injury, any epileptic equivalent, or any type of convulsion except possibly in infancy, and then not after the age of two years.

Second.—The predisposition to and the development of convulsive attacks brought about by alcoholism, syphilis, hysteria, eclampsia and other convulsion producing conditions, must be ruled out; for in the event of injury to persons so predisposed the traumatism often serves but as a precipitating factor to bring to light the condition of epilepsy theretofore latent, or as Gowers aptly remarks: "The injury is but the spark that explodes the powder."

Third.—The injury itself must have been of such intensity as to produce at least momentary loss of consciousness, and evidence of injury to the skull, spine and body should be apparent, or a perfectly clear and reliable history should be established.

Fourth.—The disease should have developed within the period of a year from the date of the injury, except in very rare cases. The more remote the date of injury, the more unlikely it is that the case is one of traumatism. The factor of time is the one that is least certain.

With these conditions of the postulate established and the so-called idiopathic or symptomatic epilepsy ruled out, the diagnosis of traumatic epilepsy is justified.

For purposes of description, traumatic epilepsy may be classified into four types, to wit:

Traumatic epilepsy of reflex origin,
Jacksonian epilepsy,
General epilepsy,
Mixed forms of the foregoing types.

TRAUMATIC EPILEPSY OF REFLEX ORIGIN

The term "traumatic reflex epilepsy" is applied to that class of cases in which there are periodic convulsive attacks due to the results of peripheral injuries. This type is extremely rare and practically the only statistics concerning it have been obtained from the records of the Franco-Prussian war and from the personal records of observing practitioners and surgeons. Siemerling states, in writing of such cases, that they occur as the result of painful and adherent scars, particularly in the region of the head and neck. He reports cures in such cases by the removal of the scars. The convulsive attacks

of this type are said to be general, and they are often preceded by auræ which have their inception at the site of the scar. Surgeons and neuropsychiatrists of repute concur in Siemerling's conclusions.

JACKSONIAN EPILEPSY

The Jacksonian type, as the name indicates, has for its expression convulsive and spasmodic attacks of certain groups of muscles, the group of muscles affected corresponding to the area of cortex injured or irritated. It is usual for the Jacksonian type to become one of the mixed forms. The convulsions gradually spread from one group of muscles, or the muscles of one limb, to the body muscles in general. At first the convulsive attacks, when limited to a group of muscles or an extremity, are not attended by loss of consciousness. The spasms may be of a clonic or tonic character, or both, and last from a minute to an hour. The spasm may be attended by momentary loss of consciousness, which is usually followed by headaches and short periods of aphasia. This type of epilepsy, like general idiopathic epilepsy, is attended by a gradual process of mental deterioration and by the development of the usual epileptic character, if surgical procedure is not promptly instituted and the pathology removed. A cure by surgical intervention is by no means always effected, because the habit of periodic epileptic explosive action through the motor paths of the brain is established, or the equivalent of a tumor has been produced by the dilatation of the blood-vessels, or degeneration of the cortical cells has taken place in the region of the injury.

GENERAL TRAUMATIC EPILEPSY

This form of epilepsy appears after injury to the head or spine. There may or may not be fracture of the skull and the brain itself may show no gross signs of injury. The convulsions of this type of epilepsy are no different in their expression than those of idiopathic epilepsy. They come on at the outset of the disease, the convulsions are general, consciousness is lost, and the usual paroxysm of the grand mal type is observed. In about one-half of all cases the first attack of general epilepsy of the traumatic form, according to Bailey, develops within the first month. The disease does not always express itself by the grand mal type of convulsion, but is occasionally shown by petit mal attacks, psychotic episodes or epileptic equivalents, and impairment

of the general mental processes. It has been noted by some observers that the process of mental deterioration is more rapid in the traumatic types than in idiopathic epilepsy. Wagner states that the degeneration occurs about three times as rapidly. The psychotic episodes have as their forerunners conditions of irritability, outbursts of anger, and states of melancholy and depression. The epileptic temperament is soon developed. Sometimes there has been observed, as Kaplan has noted, the "explosive diathesis."

MIXED FORMS OF THE FOREGOING TYPES

In the mixed types of traumatic epilepsy we have the characteristics of general and focal epilepsy. Both types of symptoms may develop simultaneously, or either type may appear first. The convulsions may begin as a Jacksonian type in some group of muscles or a limb, and spread through the body in general, associated with loss of consciousness. There is a greater tendency toward the development of mental defect and psychosis in the mixed type than there is in the focal form. The injury producing this type of traumatic epilepsy is one which destroys or badly irritates the cortex at some particular point in the motor area and at the same time injures the brain in some gross manner—possibly by laceration—but the evidences of the gross injury are not always apparent.

PATHOLOGY

The pathology in traumatic epilepsy depends on the kind and severity of the injury sustained. The skull may have been fractured by a fall on the head, a blow on the head by a blunt or sharp instrument, or a bullet wound, all of which may produce depressions of the skull, splintering, the driving of fragments of bone into the brain substance, the tearing of the meninges attended by hemorrhage, the formation of exostoses, the development of cysts, the introduction of pus germs leading to abscesses, and the formation of scars which produce pressure. When the calvarium is opened we may find some of the conditions just mentioned. Often the meninges may be adherent, and growing down from them into the brain substance may be trabeculae of connective tissue. The cortical cells in the wounded area may be entirely destroyed and replaced by an increase in the connective tissue, or the nerve cells may have undergone various degrees

of degeneration. In focal epilepsy the pathological findings are more characteristic and better localized than in the general types of traumatic epilepsy. Bailey says, concerning the pathology of Jacksonian epilepsy, "If the cerebral lesion is sufficient to cause paralysis, that symptom can be very easily be understood by remembering that the injury to the cortex has injured or killed the cells which are the essential factors in voluntary motion. But why cells, whether they do or do not retain the power of causing voluntary movement, should from time to time become the seat of irritation and thus cause convulsions remains unexplained."

THE TREATMENT OF TRAUMATIC EPILEPSY

Though the diagnosis of traumatic epilepsy is definitely established, there should be instituted medical as well as surgical treatment. Great attention should be paid to the general measures of hygiene; there should be established habits of regularity of life; a moderate amount of outdoor and mildly energetic physical exercise, varying in accordance with the muscular development of the patient, should be prescribed. The bowels should receive careful attention to prevent constipation and the absorption of toxic products from the intestinal canal. The patient's mind should be diverted from consideration of himself and his trouble into avenues of interesting mental employment and general mental activities. After the performance of the indicated surgical operation the medical treatment must be thoroughly followed in order to establish a permanent cure or improvement.

SURGICAL TREATMENT

At first thought it would seem that the surgical treatment would be of a simple character, consisting in removing the source of irritation to the cortex by lifting up or removing the depressed fragments of bone, removal of foreign bodies and exostoses, cleaning out of old hemorrhagic clots, and extirpation, in rare instances, of portions of the diseased cortex.

The matter is not quite so elemental as it seems, and certain general rules and details are to be observed if success is to attend the results of the endeavor.

The surgical operation should be performed just as soon as possible after the definite diagnosis of traumatic epilepsy has been made. As

a rule it is safe to say that recovery is unlikely in those cases where the offending cortical irritation has been removed, if the disease has existed longer than two years. There are a few exceptions to this general statement. If a cyst has been slowly formed; if the blood clots have been very slowly organized into connective tissue, or there has been a slow growth of bony substance due to nature's efforts to repair the osseous tissues, there may finally, as the result of this endeavor on the part of nature to repair the injury, arise a condition equivalent to a tumor. In these cases one would be justified in operating, even though there has been a period of five years between the time of injury and the development of symptoms.

It is found at times that simply to remove a button of bone is sufficient to produce a cure. Again, excision of the cortical tissue may be practiced if the area of irritation is definitely circumscribed. Great care must be exercised in localizing this area.

Spratling states, apropos of the time of operating, that "The time to operate is at once before the seal of chronicity is set beyond the possibility of removal. The more marked the indications of heredity as a factor in causing the disease, the closer the operation should follow the trauma, for the disease in such cases very soon becomes ineradicable."

According to Oliver, "Statistics prove that operative measures should only be employed in traumatic cases in which there are localizing features."

Matthiolius states: "The cases of traumatic and Jacksonian epilepsy show a larger percentage of favorable results than observed in general epilepsy, while the death rate after operation is much greater in general epilepsy than in cases in which the seizures are localized."

L. Pierce Clark is in accord with the idea held by most of the neurologists and surgeons, and concludes: "Traumatic epileptics may be trephined when the injury is definitely proved and stands in direct causal relation and has existed not more than two years. The prognosis will then largely rest upon the degree of neurotic predisposition present. The earlier trephining is resorted to after convulsions begin, the better the prognosis."

In discussing traumatic epilepsy Roswell Park stated: "Every case must be studied as a problem by itself. The only general laws

applying are those regarding the removal of peripheral or local foci of irritation and the destruction of paths of conduction which convey disturbing impulses."

Da Costa says: "Operations for epilepsy are distinctly disappointing and rarely curative, and are indicated only in a very small proportion of cases. They frequently produce temporary benefit. They may save life, but they are not entirely free from danger, and occasionally leave the patient worse than before. The mortality, though small, is not inconsiderable. The actual number of complete recoveries is probably under five per cent. No case should be claimed to have been cured until three to five years have elapsed since operation. Even after operation medical treatment and supervision should be exercised for a long period of time."

Brewer in writing on this same subject says: "The concensus of opinion among neurologists and surgeons at present seems to be that operations are indicated only in recent cases, within one year after the injury, in cases in which general epilepsy has followed, and in cases in which the attacks are of a purely focal or Jacksonian type, especially if an injury has preceded and corresponds to the motor center presiding over the muscles which are the seat of convulsive movements. The reason that the operation does not give permanent relief in cases of long duration is explained by the theory that the epileptic habit has become established, or that cortical degeneration has taken place which relief of superficial irritation does not remove."

The percentage of cures following surgical treatment varies with the different authorities. Matthiolius reports out of 258 cases of operations for Jacksonian epilepsy that 20 per cent. were cured, though their cases had been observed for a period of three years only, and 18 others had only been observed for one year; 15 per cent. were improved; 56 per cent. were unimproved, and 13 per cent died.

Brown reported that 13 cases were cured, 9 improved and 8 unimproved, in 30 operations performed for the relief of traumatic epilepsy.

Starr operated 29 cases, with the following results: 10 cured, 6 improved, 11 unimproved, 2 died.

Keen remarks: "When traumatic epilepsy has once been established there is less chance of operative relief," and he describes in his text on surgery a case in which the cortical lesions could easily be

separated from a bullet that had been buried just under the surface. But he found that removal of the initial sources of the disturbance did not lead to a cure. He further states: "Nevertheless, in cases of traumatic epilepsy it can be definitely stated that should the attacks present any focal symptom the patient should be operated on after the first occurrence, for the likelihood of operative relief diminishes rapidly with long continuance of symptoms; further, that even though the attacks are not completely and permanently stopped by the operation, they are apt to be lessened in severity and their interval prolonged, provided the operation discloses some lesion which can be removed, as adhesions, a depressed fragment of bone, or a foreign body."

The patient whose history is cited here was operated on by the writer at the Indiana Hospital for Insane Criminals, at Michigan City, Indiana, five years ago.

CLINICAL HISTORY

The patient, white, male, thirty-five years of age, was committed, in 1916, to the Indiana State Prison for murder. Ninety days before his incarceration he attempted suicide by shooting himself in the left side of the head, above and in front of the ear. When the patient regained consciousness some five hours after the shooting, he found himself in jail with his head bandaged. The wound to his head was dressed several times by the jail physician during the period of his trial, and at the time of his commitment to prison the wound to the scalp was completely healed. During the usual entrance examination, palpation over the site of the self-inflicted wound revealed only a very slight depression, the surface of which was uneven and a little roughened. The medical records of the jail referred to the wound as superficial and unimportant, and as having been made in an attempt at suicide.

After the patient had been in the prison for a period of six weeks he came into the prison hospital requesting relief. He gave a history of having had a dizzy attack the day before, followed by twitching and jerking of the right little finger, hand, arm and leg. These paroxysms occupied about five minutes' time. Following the convulsive attack of these members, which the prisoner carefully watched, he went into a period of unconsciousness which, according to the onlookers, was attended by violent contractions and jerkings of the

body, chewing of the tongue, frothing at the mouth, with the exhibition of bloody saliva. The patient absolutely denied ever having had such attacks before in his life. He was placed in the prison hospital for a period of three weeks' observation. During this time the patient had five convulsive attacks, which always commenced by a twitching and spasm of the little finger of the right hand, spreading to the arm, leg and face. When the face muscles became involved the patient uttered the usual epileptic cry and lost consciousness. At this time the body became involved in general convulsion. The convulsive attack after loss of consciousness usually lasted about one and one-half minutes. The period of time between the first twitching of the little finger and the grand mal attack was about five minutes, and during this interval the patient talked to the hospital staff in a rational manner. The patient was again carefully examined neurologically and it was ascertained from his relatives that he had never before suffered from epilepsy. The site of the injury to the head was again examined, but revealed only a slightly roughened depression, the scar tissue of the scalp being of such a character as to interfere with determining just how much damage was done to the bone beneath. The prison hospital was not at that time equipped with satisfactory apparatus so that a skiagram might be made of the skull, and the patient could not be removed from the institution.

After the fifth attack the patient was operated. A trephine operation was performed at the site over the middle of the Rolandic area. The scalp at this point was very hard to incise, due to the presence of scar tissue. On clearing away the scalp there was revealed a Y-shaped crack in the outer table, and at the center of the arms of the Y there was a slight aperture of irregular shape, about an eighth of an inch in diameter, in which there was a foreign substance. A button of bone was removed, and there was found to be penetrating the inner table of the skull a badly misshapen and flattened twenty-two calibre bullet which had been pressing down upon the brain and its coverings. The bullet was firmly fixed in the bone and adherent to the dura. There was some evidence of slight pial hemorrhage. Great care had to be exercised to effect the separation of the roughened button of bone with its imbedded bullet, from the dura. The meninges were slightly torn in effecting the separation. The wound was surgically closed.

The patient made an uneventful recovery from the operation. Five years have elapsed since it was performed, and there has been no recurrence of any symptoms of epilepsy, and the case may be regarded as entirely cured.

CONCLUSION

The writer considers this a typical case of traumatic epilepsy, all other factors having been ruled out. The area of the cortex involved was circumscribed and very definite. The injury was recent, and therefore the period of cortical irritation was relatively short, and the epileptic habit of explosion had not been firmly established. With the prompt removal of the foreign substance a complete cure was effected.

EOSINOPHILIC PLEURISY *

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THE paucity of reports in the literature dealing with pleural effusions containing a large number of eosinophiles is the reason for reporting in detail two cases which have been followed for a period of five years.

The first report of pleural effusions of this nature was that of Harmsen.¹ Both cases reported by him had an hemorrhagic effusion, the first of insidious onset, the second as a sequelum of stab wounds of the thorax. In both cases the right side was involved; early thoracentesis yielded a relatively low percentage of eosinophiles later rising to 39 per cent. and 79 per cent. respectively. His second case showed a moderate eosinophilia in the peripheral blood, amounting to 9 per cent. thirty-four days after the injury. Klein,² under whose direction Harmsen reported his cases, added another case and drew the conclusion from these three cases that the eosinophiles were leucocytes that had ingested hemoglobin and that this accounted for the late increase in eosinophiles in the peripheral blood and in the pleural fluid. In Klein's case the first count in the blood was 12.5 per cent. gradually rising to 40 per cent. Both thoracenteses which he reported contained about 75 per cent. eosinophiles. His patient had a hemorrhagic effusion as a part of a general sepsis following an operation for hemorrhoids. Autopsy showed a beginning effusion of the left pleural cavity as well as the right but offered no further data as to the nature of this process. Independently of these two reports, Widal and Ravaut³ described this condition, mentioning in the course of another study that they had observed three cases, two of which were tuberculous. One of the latter had been in the tropics and showed an enlarged liver and general adenopathy. Curiously enough this patient showed the smallest eosinophilia of these cases.

* From the Medical Clinic of the Peter Bent Brigham Hospital, Boston, Mass.

¹William Harmsen, Inaug. Diss., Dorpat, 1894.

²Klein, S., *Centr. f. Med.*, 1899, vol. xx, p. 97.

³F. Widal and Ravaut, *C. R. Soc. Biol.*, Paris, 1900, vol. lxi, p. 648.

Of the remaining two cases, one was frankly tuberculous and the other post-typhoidal. They note that the fluid from these cases was unusually toxic. Guinea pigs given 20 c.c. intraperitoneally died in a few days of a fulminating peritonitis in which no organisms could be demonstrated. Of the pigs given 12 c.c., only the one injected with the fluid from the typhoid patient lived and this animal showed no evidence of tuberculosis when killed. These are the only detailed reports that we have found in the literature, although the condition is mentioned in most text-books of diseases of the chest. Norris and Landis comment on the frequency of hemorrhagic effusion in this type of pleurisy.

Brief histories of our cases follow:

J. L. L., white, male, age 24, was admitted to the Surgical Service of the Brigham Hospital (Surg. No. 5469) on September 27, 1916, with the complaint of pain in the right back and in the front of the abdomen. His family history and habits are not of importance. He is single and his occupation is that of a mechanic in an automobile garage. Past history: He apparently had an attack of pleurisy a few years before; further details were not obtainable from the patient. His best weight was 146 pounds a year and a half ago; present weight 136 pounds. The patient dates his present illness from three days ago, at which time he was seized with a rather sharp pain in the back of his right chest. About four hours after the pain began, it shifted around to the front of the abdomen and finally seemed to localize in the right lower quadrant, gradually increasing in intensity and being most severe when he took a deep breath. It was severe enough to make the patient stop work and go to bed.

He has had no nausea, vomiting, or gastric disturbances, although his bowels have not moved for two days. There have been no urinary disturbances; no swelling or tenderness in the abdomen. The patient states that the attack of pleurisy two years ago was precisely similar to this one.

On physical examination the patient is a fairly well-developed and well-nourished young man lying quietly in bed. The skin is moist and hot but shows no eruptions or other lesions. The head, mouth, and pharynx are negative. The thorax is well-formed, expansion is good and equal on both sides. Over the right back from the angle of the scapula extending around to the front of the chest,

there is almost flatness on percussion. Over this area the breath sounds are slightly diminished and expiration is slightly prolonged. The voice sounds over this area are bronchial in character. Just above this dull area there is a slight hyper-resonance and the left chest is hyper-resonant throughout. No râles are heard. Kroenig's isthmus is not narrowed. No succussion sound is made out, no metallic tinkling and no coin sound.

The apex impulse is not seen, but is felt 1 cm. outside the nipple line on the left. There is no precordial bulging, and no thrill. The relative cardiac dullness corresponds to the P. M. I., 9.5 cm. to the left of the mid-sternal line, and is apparently at the right sternal margin on the right. It extends upward into the second space. Both sounds at the apex are clear and regular. The pulmonic second sound is greater than the aortic. No murmurs are heard. The pulses are equal, regular synchronous, of good volume and tension. Blood-pressure: Systolic 110, diastolic 90. The abdomen is quite negative, except for slight pain on deep palpation over the right lower quadrant. Rectal examination was negative. Aspiration was done twice. Bloody fluid was obtained both times.

On September 28th, patient was transferred to the Medical Service (Med. No. 5368). On September 29th, it is noted that the right chest in front is flattened and expansion is not as good as on the left and the intercostal spaces in the lower half and in the axilla are full when the patient is in a sitting position. Over the right chest tactile fremitus is somewhat diminished over the upper part and present over the lower when the patient is in a sitting position. There is good resonance over the right apex from the right clavicle to the fourth space below which there is dullness merging with liver dullness. Over this area breath sounds are distant but rather bronchial in type. Similar signs are found from the mid-scapula to the base behind, except at the extreme base, where there is a definite bronchial breathing and egophony. No râles are heard. The dullness in the back does not shift with change of position. It extends through the axilla to the anterior axillary line, but does not go beyond this. About the region of the anterior axillary line and the lower part of the chest in front the dullness and flatness shifts with change of position. In the right axilla may be heard scratchy sounds suggestive of a friction rub. The character of the resonance over the front of the chest

and especially over the right of the chest when the patient is lying down suggests air as well as fluid in the pleural cavity. There is no Grocco sign. Patient's temperature is 101. By October 6th, his temperature remains about 99, pulse averages 70, respiration, 20. On September 29, thoracentesis of 430 c.c. was performed and bloody fluid obtained. Since then patient's signs have changed but little. However, by October 6th they seem to be somewhat less. White count is 8400. Fluid obtained from the chest did not coagulate. Specific gravity, 1014. Albumin 3.4 per cent. Guinea pig gave no evidence of tuberculosis. On October 7th, the signs of re-accumulation of fluid have become marked, but it was thought best not to tap. X-ray examination showed presence of fluid. On October 17th patient showed no change in signs. Grocco's sign had become positive. The amount of fluid has not increased. On October 21st patient's temperature showed a tendency to rise and on the 22nd he was put back to bed. He continued to have pain in the side, signs had slightly increased and the evening temperature of the 22nd was 102. On the 22nd 1300 c.c. of bloody fluid were withdrawn. Just before this his white blood count was 13,600. Smear of the chest fluid showed a marked eosinophilia. This fluid showed 367,000 red cells, 4000 white cells, hemoglobin 6 per cent., albumin 4 per cent., cultures negative. Guinea pig negative for tuberculosis. Differential: Neutrophils 3 per cent., lymphocytes 19 per cent., eosinophiles 77 per cent., Mast cells 1 per cent. On October 23rd his white count had dropped to 10,000 and the signs had diminished. The X-ray examination at this time showed evidence of thickening of the pleura in the lateral aspect of the chest but no evidence of consolidation. Costophrenic angle was clear. The patient continued to improve, his temperature gradually became normal. No evidence was found of intrapulmonary process. Urine was negative. Hemoglobin on entrance was 60 per cent. Red blood count 2,940,000. White blood count 7280. The smear at this time showed 1 per cent. eosinophiles. On October 23rd hemoglobin had risen to 90 per cent. with a white blood count of 10,000. Smear showed 4 per cent. eosinophiles. Wassermann and stool were negative. Patient was discharged apparently well on November 24, 1916. A year later he returned to the Out-door Department and showed no traces of his former pleurisy on examination. We heard from him in February, 1921. He is working outside of Boston,

has been perfectly well without complaints of any kind since his discharge.

E. J. F. (Med. No. 5382), white, male, married, age 48, was admitted to the Surgical Service on August 18, 1916. Discharged on September 9, 1916. Re-admitted to the Medical Service on October 1, 1916. On the Surgical Service he was admitted with the diagnosis of acute cholecystitis and cholelithiasis. Cholecystectomy was done on August 18th and the patient made an uneventful recovery. Family and past histories are of no importance save for evidence of chronic cholecystitis. Best weight 178 pounds, twenty-five years ago, present weight 156 pounds. Since patient left the Surgical Service he has been slightly bothered by shortness of breath and pain in the lower right chest on taking a deep breath. He has no cough or night sweats, but returns because of this discomfort. He believes that this began just before he left the Surgical Service. One day he had a bath in bed in a corner of the ward and was chilled, he believes, by an electric fan which was necessary on account of the heat. Soon after he was discharged and a few days later this pain came on suddenly. His pain increased at first and then grew less. He has been slightly short of breath and has a heavy feeling in the right chest. Physical examination shows a well-developed fairly well-nourished man of large frame lying quietly in bed with no apparent discomfort. Skin is clear. Respiration quiet and deep. Head is negative. There is no adenopathy. Thorax is well-developed. Expansion is much better on the left and at the end of expiration the right chest seems slightly more bulging, than the right. Heart: Left border of cardiac dullness is in the sixth space, 11.5 cm. to the left of the mid-sternal line and upper border is in the third space. It is not enlarged to the right where cardiac dullness is apparently continuous with the dullness of the lower chest. Apex impulse neither seen nor felt. Action is regular and slow. Sounds are of good quality, no murmurs heard. Retrosternal dullness is not increased. Vessel walls are not palpable. Pulses are of good quality. Blood-pressure, systolic 120, diastolic 80. Lungs: The left lung appears to be clear. When the patient is sitting up there is a good resonance over the upper part of the right chest, dullness below the third space in front. On lying down the dullness begins in the fourth space. This dullness quickly changes to flatness, over which breath sounds

are diminished and then absent. No râles are heard. Posteriorly there is resonance at the right apex. From about two fingers' breadth below the angle of the scapula posteriorly there is at first dullness which quickly changes to flatness which extends to the right base around through the right axilla with the same signs as in front. Over this area tactile fremitus is absent, breath sounds are diminished or absent and just above it there are a few moist râles. A few moist coarse râles can be heard between the spine of the scapula and the vertebral column in the large bronchi. These change quickly with respiration, but do not entirely disappear. There is slight tenderness over the right costal margin in front and over the scar of the cholecystectomy operation of six weeks ago. The abdomen is negative. On October 3rd, patient was tapped and 1300 c.c. of clear straw-colored fluid withdrawn. Albumin 5.5 per cent., smear showed polynuclears 3 per cent., small lymphocytes 61 per cent., endothelial also 1 per cent., eosinophiles 19 per cent., cultures and pig were negative. On October 7th no pleural friction could be heard but a curious resonance developed over the right back just below and to the outside of the angle of the scapula where the note suggests Skodaic tympany. No râles are heard although breath sounds are somewhat diminished and voice seems distant. Tactile fremitus is diminished also over the right back. The curious resonant note is probably caused by air in the chest. This was not noticed, however, immediately after tapping although the air was probably admitted at this time. Patient feels well. X-ray shows partial pneumo-thorax and a partially collapsed lung. Patient is running no temperature and wishes to go home and so is discharged on October 7th. Urine is negative. Wassermann is negative. Blood on October 1st showed hemoglobin 72 per cent. (Sahli). Red blood count 3,600,000. White blood count 13,200. Smear shows polynuclears 60 per cent., lymphocytes 30 per cent., large mononuclears 5 per cent., eosinophiles 5 per cent. The patient continued an uneventful convalescence. In a letter of March 18, 1921, the patient describes himself as quite without further symptoms referable to his pleurisy since his discharge from the hospital. An X-ray, taken September 29, 1921, shows slight pleural thickening on the right, but is otherwise normal.

These two cases of pleural eosinophilia form about 1 per cent. of the cases of pleurisy with effusion seen in this clinic in the past

eight years. It is of interest to note that of the eight cases reported in more or less detail only two are of proved tuberculous origin. The fact that one occurred in the course of typhoid fever and one incidental to an operation for cholelithiasis seems an interesting though hardly significant factor. Neither of our cases showed a marked eosinophilia in the blood, though in both cases the percentage of eosinophiles was increased. There was no evidence that either had any parasitic disease; the one lived all his life in the United States while the other though born in Germany has never been out of the north temperate zone.

It does not seem justifiable to speculate about the possible etiology of such an exudate except to point out that Klein's explanation of this phenomenon will not suffice inasmuch as our second case did not have a sanguinous effusion. It does seem that this condition deserves further study in relation to other types of pleural exudates.

REPORT OF THREE CASES OF AMENORRHŒA OCCUR- RING IN INSANE PATIENTS AND CLEARING UP UNDER GLANDULAR THERAPY

By L. D. HUBBARD, M. D.

St. Elizabeth's Hospital, Washington, D. C.

THESE three cases are presented, not with the idea of proving any hypothesis concerning the treatment of endocrine disturbances, but merely to provide additional data on a subject still in its experimental stages. The women studied were all patients in St. Elizabeth's Hospital (Government Hospital for the Insane), Washington, and were diagnosed dementia precox. I shall give a brief report of each one, touching upon the history, the treatment, the physical results and the mental condition at the time of writing (July, 1921).

The first case is that of a woman twenty-three years old on admission to this hospital March 13, 1915. Two brothers were alcoholic. The family history was otherwise negative. Her birth and early development were normal. Puberty occurred at the age of fourteen and menses were bi-weekly. At twenty-one she married and one year later had an instrumental delivery with severe cervical lacerations. Although the child appeared normal it lived only nine days. The patient had frequent hemorrhages and after four months she was treated with tampons. She became pregnant again and had a normal delivery in the hospital February 27, 1915. Eight days later she developed a psychosis, was taken to a private sanitarium, and on the thirteenth of March was brought here. Her condition on admission was such that complete examination was impossible. No gross abnormalities were discovered and a pelvic examination was not done. Laboratory examinations were negative. For about four years she continued excited, was assaultive, destructive, attempted to run away and masturbated excessively. During this time she never menstruated. In the summer of 1919, however, it became apparent that the degree of excitement varied at different times, in a monthly cycle—two weeks extreme excitement and two weeks more normal behavior. In the fall of that year psychoanalytic treatment was begun and there

was marked improvement. A gynecological examination done about this time showed no gross pathological condition and on January 22, 1920, she was put on glandular therapy, receiving ten grains of corpus luteum preparation in tablet form three times a day. A month later (February 19, 1920) the dosage was changed to five grains four times a day but as there were no apparent results this amount was doubled on the fourth of March. On the thirteenth of March, while at the height of an excited period, she picked plaster from the wall and inserted it in her vagina. For two days she menstruated freely, for the first time in five years. Medication was continued and on April 16 there was a very slight flow of blood. This occurred again on the nineteenth. A normal three-day period began April 27, during a quiet interval. Medication was decreased on May 6, 1920, and she received five grains three times a day. The next period occurred May 30 and the next June 30. A suggestion of reversion to the interval of her girlhood then appeared for she menstruated July 17. A series of normal intervals followed and then a shortening again: August 14, September 12, October 12, November 7 and December 1. From this time to date the notes show that she has menstruated regularly although the medication was discontinued in July, 1920. Her mental condition has improved markedly and she has had only one severe excitement since December, 1920, but the improvement was very noticeable for some time before organotherapy was instituted and she received psychoanalytic treatment coincidentally.

The second case is that of a single colored woman who was admitted to this institution October 12, 1918, at the age of thirty-six. She gave a history of having been a very delicate child, suffering with frequent attacks of "grippe" and "sore eyes." Her psychosis had its onset two years before admission, subsequent to the death of her mother to whom she had always been devoted. Date of puberty is not recorded but the patient states that she did not menstruate for a couple of years before coming here. On admission she was quiet and indifferent, had vague delusions of persecution. She weighed ninety pounds and was five feet, three inches tall. Pubic and axillary hair were abundant and there was hypertrichosis of nipples and chin. Mammæ were undeveloped but nipples were large and erect. Gynecological examination showed elongation of the left labium minor and darkening of the mucous membranes. Hymen was unruptured,

uterus was small but in good position, cervix hard and long. All other examinations were negative. According to the patient's statement she menstruated slightly once during her first few months here but this is not substantiated by nurses' or physicians' notes. Her physical condition improved until February 15, 1920, when she developed a cough and was suspected of having an incipient pulmonary tuberculosis. In July, 1920, she was found to be very anemic. An X-ray of the lungs showed an old healed lesion and the cough had disappeared. In August it was discovered that she was trying to starve herself. She weighed 78 pounds, had tremors of extremities and face, pulse rate 120, very feeble, thyroid enlarged. She was tube fed twice daily and gained slowly, reaching her greatest weight, 101 pounds, in January, 1921. On November 1, 1920, organotherapy was begun. On the advice of Dr. N. D. C. Lewis of the pathology department of this Hospital, who kindly examined the patient and supplied the dried glandular substance which was used, she was given one grain of adrenal substance and one grain of ovarian substance three times a day. As it had to be given with the liquid through the nasal tube, it is probable that she never received the full dosage. She weighed ninety pounds at this time. Mentally she was full of delusions of persecution of a sexual character and she had both auditory and visual hallucinations. She would never explain why she would not eat. Two weeks later her dosage was increased so that she was given six grains of each of the gland substances per day. On January 30, 1921, she menstruated—her first free menstrual period for four years. Medication was continued and another period occurred February 18. The next period was on the fourteenth of March and one more occurred in April. For three weeks in April and May she ate but refused medication. She ceased eating as suddenly as she began, however, and for the past two months has been given her powders, but there has been no menstrual flow since April.¹ Her weight has fallen again to ninety-three pounds and her mental condition shows no improvement.

The third case is that of a married colored woman who was admitted here February 13, 1920, in her twenty-fourth year. She

¹ Since this manuscript was submitted for publication patient has begun to menstruate regularly.

developed normally and was a healthy, bright child. Puberty occurred at thirteen and she menstruated freely at twenty-eight day intervals. When she was seventeen she married and had two full-term deliveries. A miscarriage occurred in March, 1919, and she suffered with abdominal pains and fever for three weeks. The psychosis apparently had an insidious onset during the few weeks following this illness and she became gradually worse until she was so irritable and assaultive that it was necessary to hospitalize her. On admission she weighed one hundred and two pounds and was four feet, eight inches tall. Thyroid was palpable and symmetrically enlarged. There was a fine tremor of the tongue. Gynecological examination showed reddened mucous membranes and considerable leucorrhœa. There were old lacerations of the cervix and the uterus was retroverted. Up to the time of admission to the hospital the patient had menstruated regularly. Laboratory examinations were negative. She gained weight but suffered with obstinate constipation. February 16, 1921, she was put on glandular therapy under the direction of Doctor Lewis. She received two grains each of the ovarian and adrenal substances mixed with her food once a day. This method of administration made the dosage uncertain but she refused capsules. On the eleventh of March the dose was given twice a day and on the twentieth of April it was increased to three times a day. She menstruated on July 13, 1921, for the first time in seventeen months.

These patients had periods of amenorrhœa of five years, four years and seventeen months respectively. They received glandular treatment for two months, three months and five months before the menses reappeared. The first case received corpus luteum tablets in increasing doses, the second and third received dried ovarian and adrenal substances in powder form. Both these cases showed symptoms of hyperthyroidism. The number of cases reported is too small to prove that the reappearance of the menstrual flow was the result of the glandular therapy but it seems probable that it was, although spontaneous relief of long-standing amenorrhœa occurs. In the first case there was mental improvement but it began before organotherapy was instituted and seemed to be dependent on the psychoanalytic treatment. No lasting improvement occurred in the second case and the third has shown no change to date.

DUODENAL ULCER WITH PORTAL VEIN COMPRESSION AND ASCITES

By PROFESSOR HANS FINSTERER*

AFTER Mayo in America and Moynihan in England had pointed out the frequency and importance of the duodenal ulcer, our knowledge of this affection has increased greatly of late years, due to its operative treatment and the results thereof.

Because of certain periodical recurrence of the ulcer with long remissions of comparatively little trouble, it may in case of a sudden aggravation lead to the greatly feared complications of acute hemorrhage and perforation into the open abdominal cavity, two occurrences which are unfortunately not rare. It is likewise a well-known fact that the ulcer—owing to the long duration of the disease—may extend to the surrounding organs and after penetration into the neighboring viscera (pancreas, liver and gall-bladder) may lead to the formation of large tumors which, because of their size, may be mistaken for a neoplasm of the pylorus. By spread to the ligamentum hepatoduodenale serious difficulties arise in the way of a radical operation so that this must be avoided in the interest of the patient.

But it is certainly a very rare occurrence that it leads to the formation of a severe ascites by extending to the ligamentum hepatoduodenale, which in connection with the emaciation might all too easily be interpreted as the consequence of a malignant degeneration of a long existing ulcer. A case of this type operated on by me some time ago presented the following facts.

The patient was a man forty-one years old. He had had stomach trouble for fifteen years and had been treated for overacidity. He had pains for some hours after meals, but often also when the stomach was empty. This pain was relieved after eating. He suffered from sour belching and sometimes vomited sour fluid. Repeated treatment brought temporary relief, but even then he could not take any acid food. During the war his inability to obtain a proper diet greatly aggravated the trouble. The pain after eating increased and he vomited acid fluid up to a quantity of two

*Vienna, xix Vegagasse 15.

liters and his stools were frequently black and repeatedly bloody. He had lost sixty-seven pounds in weight when he came to operation.

His condition was as follows: Tall, very thin, pale and almost cachectic. Weight, 114 pounds; abdomen distended, particularly in the upper part; stomach dilated down to two fingers' width below the navel; liver dullness not increased; movable fluid demonstrable in the abdomen; pressure point to the right of and above the navel; slight tension of the right rectus muscle.

The Röntgen picture showed that the stomach was dilated, moved far to the right and reached down to two fingers' breadth below the navel; peristalsis was increased. Repeated examinations of the gastric juice always showed hyperacidity, total acidity seventy to ninety, free hydrochloric acid sixty.

The operation was performed under local anæsthesia with the help of ether. A large amount of clear ascitic fluid was evacuated through the median incision. Over the dilated and hypertrophied stomach the veins were tensely filled to the thickness of a thumb; they were present principally on the large and small curvature. The veins in the large omentum were greatly dilated, as were also the veins in the mesentery of the uppermost jejunum loop; those in the mesocolon less. The liver itself had a perfectly normal appearance; there was no increase of consistency, no enlargement and the surface was smooth. The gall-bladder adhered to the duodenum and the colon. After the adhesions of the colon were loosened it showed that the small omentum was callous and œdematous. The entire upper portion of the duodenum was occupied by a large ulcer, which reached outward from the pylorus to the descending crus, continued into the ligamentum hepatoduodenale and reached the liver porta. Here the penetrating ulcer extended on the one side, from the gall-bladder, and on the other side from the ligamentum hepatoduodenale. The gall-bladder itself, although thickened, was free from stones. The ulcer penetrated backward into the pancreas, the head of which was strikingly hardened. As the ulcer reached outward to the papilla, a resection was impossible even if the base of the ulcer was left. The stomach was therefore severed three centimeters distant from the pylorus in order to exclude the large ulcer and the aboral end was closed by two suture layers. Then two-thirds of the dilated stomach were resected to an extent of fifteen centimeters on the small curvature

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and twenty-five centimeters on the large curvature. The remainder was closed in its upper portion and the lower portion was united end to side with the first jejunum loop in the usual manner; complete abdominal suture followed.

The after-course was uneventful. The ascites did not return and after four weeks the patient was discharged cured.

He convalesced quickly and gained fifty pounds. Every kind of food is taken without trouble, even acids. Examination of the stomach shows the total acidity eight, free hydrochloric acid negative, and abundant gall in the stomach contents.

The appearance of ascites with duodenal ulcer may be explained by compression of the portal vein as the result of inflammatory tissue. As in the case of exclusion, the ulcer rapidly retrogresses, it is plain that the compression of the portal vein ceases and that the ascites does not return again. Another explanation of the appearance of ascites is offered by a thrombophlebitis of the vena portæ due to the direct spread of the inflammation. This is a rare occurrence. I recall the case of a man, fifty-four years old, in whom an exploratory laparotomy was made on account of cancer. At the autopsy an ulcer on the small curvature with erosion of the vena coronaria and a portal vein thrombosis was found to be the cause of the anæmia. It is not stated in the history of this case whether ascites was present or not, but it seems probable. In our case I would exclude the thrombophlebitis because otherwise so uncomplicated a course after the operation would scarcely have happened.

The appearance of ascites with duodenal ulcer is extremely rare. In a number of cases of duodenal ulcer operated by me I found only one other accompanied by ascites of any great amount and in this there was an associated cirrhosis of the liver.

Knowledge of the fact that a duodenal ulcer may cause an abundant ascites is particularly important because in the case of stomach trouble of many years standing accompanied with emaciation, and in spite of hyperacidity, we may suspect a carcinomatous degeneration of the chronic gastric or duodenal ulcer. In view of the age, appearance and emaciation of our case, and despite the history and previous findings which spoke for a duodenal ulcer we would have been justified in suspecting a malign degeneration, rarely as this occurs in the duodenum.

My contention in a case of this kind is that an exploratory operation should be done even if the chances are all in favor of malignancy as it may prove to be a simple ulcer. A similar significance attaches to ascites when it is attributed to a simultaneous liver cirrhosis, for the demonstration thereof suffices to decline every stomach operation on account of the danger of hemorrhage.

The spread of the duodenal ulcer to the liver porta may also gain in significance by the fact that in consequence of erosion of the vena portæ or the arteria hepatica it may lead to a fatal hemorrhage.

The surgical treatment may be greatly influenced by the seat and extent of the ulcer. In my opinion resection of the duodenum with the ulcer should be performed, and only if this be impossible to exclude the ulcer. It is clear that in the spread of the ulcer to the ligamentum hepatoduodenale the resection of the duodenum is possible only if the ulcer bottom is left behind. If, however, as in our case, the ulcer reaches outward to the papilla the operation must be declined as too dangerous. But it is not necessary to endanger the life of the patient by such a complicated operation as even after exclusion, very large ulcers of the duodenum may retrogress in shortest space of time, and the patients remain permanently free from trouble if half the stomach had been likewise removed. Since seeing an instance of this rapid disappearance of a very large ulcer at the autopsy made two months afterward, I have become somewhat more reserved as regards the duodenal resection in favor of exclusion.

In this case we had to deal with a patient fifty-one years old in whom a large ulcer extended to the gall-bladder and ligamentum hepatoduodenale and penetrated into the pancreas. This ulcer reached outward to the papilla and extended up to the liver porta and its base was partly formed of the gall-bladder. As a resection was almost impossible, half of the stomach was resected in order to exclude the ulcer and permanently remove the hyperacidity. After an ideal course and rapid improvement, fever, chills and pain in the liver region appeared five weeks afterward, the cause of which was assumed to be liver abscesses after cholangitis. Another operation, cholecystostomy, brought no healing. At the autopsy, seven weeks after the first operation, multiple liver abscesses were found and besides this a stone wedged into the cystic duct. The large ulcer of the duodenum had completely retrogressed, and only the fixation

of the duodenal mucous membrane to the gall-bladder and the pancreas corresponding to the original spot of penetration could be demonstrated. Therefore, in this case one of the largest duodenal ulcers I had ever seen had completely healed in an incredibly short time by simple exclusion.

The fact is of great diagnostic and prognostic importance that ascites may appear in consequence of compression of the portal veins even in a large ulcer of the duodenum or of the pylorus of long standing, without malignant degeneration. The knowledge of this fact is important for the reason that the exploratory laparotomy—which would show the true state of affairs—may not be declined on the assumption of a carcinomatous ascites in consequence of malignant degeneration of the ulcer.

ENDOTHELIOMA OF PLEURA SIMULATING SPINAL CORD TUMOR

By WALTER FREEMAN, M. D.

PRESENTATION OF A CASE FROM THE SERVICES OF
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SUMMARY.—Pain, anæsthesia and muscular wasting in the distribution of the 1st, 2nd, 3rd, and 4th thoracic segments, sympathetic phenomena. Few symptoms of cord compression. Exploratory laminectomy without result. Later finding by X-ray of tumor destroying second and third ribs.

The patient is fifty-three years old, a travelling salesman by occupation. His chief complaint is pain in the left axilla of a burning, tearing nature, keeping him awake at night, and preventing him from conducting his business.

The history dates back about eleven months. Coming at first intermittently but later constantly, and situated high in the axilla the pain is described as of a burning or tearing quality, as though the muscles were being torn from the bone. At first he was satisfied with the usual anti-rheumatic treatment, but with the increase of the symptoms he consulted a physician in New York, without, however, obtaining any true insight into his condition. It was not long before the pain which was at first confined to the under surface of the arm and axilla, and "deep" in the shoulder, extended down to the elbow, over the front of the chest, and into the scapular region, always on the left side. The pain in the chest and the radiation down the arm suggested the diagnosis of angina pectoris, but the heart and vessels were apparently in good condition.

He entered the sanitarium at Battle Creek for extensive study. Physical examination of the shoulder was negative. The heart was normal; blood-pressure, 140 systolic; pulse, persistently above 80. Urine, blood count, Wassermann reaction, and X-ray plates of the head, chest, spine and shoulder were all negative. There was no free

hydrochloric acid in the gastric contents. The patient returned to New York unimproved.

Early in August a patch of hyperesthesia was discovered in the axilla, and later, according to the patient's nephew who is a physician, there was an area of anæsthesia within this patch. This was the first objective neurological sign. A chart prepared by Déjerine (Fig. 1.) shows the distribution of the sensory fibres of the spinal segments on the under surface of the arm. It is seen that the 8th cervical segment is distributed down as far as the ulnar side of the ring finger, the 1st thoracic to the wrist with some overlapping on the ulnar side of the hand, that the 2nd thoracic extends down nearly to the elbow, whereas the 3rd thoracic supplies but little more than the axilla. The 4th thoracic segment supplies the lower portion of the axilla and a band about the chest an inch or so above the nipple. The 5th segment includes the nipple. Higher up on the chest, the distributions of the 2nd thoracic and 4th cervical are in immediate contact, so that whereas in other areas there is a shading of sensibility, here the line of demarcation is very sharp. There is no overlapping of the sensory fibres. Doctor Spiller has verified the accuracy of the diagram in a series of cord tumor cases in which for the extraction of the tumor it was necessary to sacrifice the dorsal root.

The objective disturbance of sensation was thus confined at this time to the third thoracic segment, whereas the pain was felt in the first to the fourth segment distribution.

About the first of August, nine months after the onset of the symptoms the burning pain extended down as far as the wrist, and the patient noticed fibrillary twitchings in the muscles, an increasing weakness of the grip of the left hand, and also an atrophy of the hand. The whole limb showed wasting, possibly from disuse. The flesh of the finger-tips fell away from the nails, and there was difficulty in the apposition of the thumb and the little finger. The first thoracic root was thus becoming involved in the process.

Finally on August 27th or 28th the patient noted that the left pupil was smaller than the right, and on examination two days later, Dr. Joseph Collins, of New York, definitely determined a sympathetic syndrome on that side, enophthalmus, narrowing of the palpebral fissure, and constriction of the pupil. Spinal fluid examination was entirely negative.

Statistics of spinal cord tumors, giving the incidence at various levels, show that the great majority of them are located in the lower cervical and upper thoracic region, and many of them present this sign of sympathetic involvement. The syndrome named after Mme. Déjerine-Klumpke shows definitely that the lesion is within a centimeter or two of the spinal cord, for the sympathetic root is given off very soon after the emergence of the root from the vertebral canal, and while the narrow palpebral fissure and small pupil are seen in brachial plexus injuries, they are seen only when the lesion is very high and very close to the cord. In association with a slowly developing segmental type of pain, anæsthesia and muscular wasting, the sign is almost pathognomonic. The diagnosis of spinal cord tumor or of one near the vertebral column is warranted.

At this same examination Doctor Collins found a mass in the supraclavicular fossa on the left side, rounded, firm and not attached to the skin. The finding of this mass was corroborated by at least five other physicians, so there can be no doubt about its having been present, but yet, one week after their examination the mass had disappeared, and no explanation could be advanced as to its nature. On the 30th of August, an X-ray plate of the spine and shoulder was pronounced negative.

Other than the pain in the shoulder and weakness in the arm the patient has no complaints. He has always been healthy, denies venereal disease, has a good inheritance, and his habits are excellent.

Physical Examination (Sept. 8, 1921).—A rather thin Jewish male of 53. He carries his left arm away from his body, with his hand in the pocket of his coat. The face has a slightly drawn expression. He stands erect and walks without difficulty, not limping or dragging the foot. No Romberg sign.

The head is well formed, nose and ears negative, a plate in the upper jaw, a few good teeth in the lower. Tongue protruded in the median line without tremor.

Eyes.—The left palpebral fissure is slightly narrower than the right, and the left pupil is distinctly smaller. Both pupils react to light and accommodation. No ptosis or extraocular palsy. Vision good, visual fields normal.

Neck thin, submaxillary glands palpable. No enlarged lymph-nodes, no masses, thyroid not palpable.

Thorax.—The supraclavicular fossæ are normal, and symmetrical, no mass palpable. From the base of the neck down almost to the nipple line on the left side anteriorly and posteriorly, and extending down the arm, the skin is dry and warm to the touch, as distinguished from the normal cool moist feel of the skin on the opposite side. No pilomotor response is obtained in this area. Sweating is fairly profuse, but limited to the right side of the chest. Heart and lungs are negative. Abdomen and genitalia negative.

Extremities.—The right arm is normal in all respects. The skin of the left arm is warm and dry, and back of the finger-nails it is shiny. The skin of the palm is thin and red. When the arm is allowed to hang down for a few moments it assumes a cyanotic tint. The biceps and triceps muscles are weak but not markedly so. The girth of the upper arm is less than that on the opposite side, and there is still a greater discrepancy in the girths of the two forearms. The grip is very weak, and the fingers cannot be held closed with any force. There is some difficulty in the apposition of the thumb and little finger. There is a wasting of the whole limb including the thenar and hypothenar eminences, and the interosseous muscles. The biceps tendon reflex is present in normal strength. The triceps is not obtained, nor are the radio-carpal or extensor reflexes. Percussion of the triceps muscle yields a sharp localized contraction, and once during the examination, this muscle was seen to twitch.

On the right side all reflexes are obtained in a normal manner.

The lower limbs are normal in girth, show no wasting, and muscular power is good. The patellar and Achilles reflexes are very prompt, possibly a little more so on the left. There is no ankle or patellar clonus, and no Babinski reflex. Coördinative tests are well performed.

Sensation.—The disturbances are shown in the photographs (Fig. 2), the heavy line enclosing the area of analgesia, and the shaded area representing the anæsthesia. Sensation to heat and cold is lost on the ulnar side of the arm down to the wrist, and for a larger area surrounding the axilla. There is also an extension of the thermanæsthesia to the midline of the body in a narrow strip just above the level of the nipple, the sensation to heat being lost over a broader band than the sensation to cold.

Wishing to have all the facts at our command, we asked Doctor

Pancoast to examine the X-ray plates which the patient had brought with him from New York. He pronounced them negative.

No sign of any mass in the supraclavicular region was found, and even to this present time the nature of the mass remains obscure although the reason for its appearance is now a little more obvious.

(At this point the patient was taken from the clinic.)

The patient was prepared for operation, and the spine of the 1st thoracic vertebra was marked with silver nitrate. No further X-ray plates were made although Doctor Frazier usually likes to have an X-ray to determine the exact level of the silver nitrate mark with respect to the spines of the vertebræ.

Pre-operative Diagnosis.—Tumor of the spinal meninges involving the second and third thoracic roots; but the possibility of an extravertebral lesion was recognized. The symptoms seemed sufficiently definite to justify operation as it gave the patient the only chance of his life.

In the cervical region, and also in the upper thoracic, the spinal roots emerge from their foramina soon after leaving the cord, running only a comparatively short intraspinal course. In the lower thoracic and lumbar regions the roots descend within the canal for some distance before making their exit through the intervertebral foramina. Thus a very small tumor at one of these lower levels while springing from a single root may press upon and give symptoms of involvement of several roots. It is important therefore to determine the root first to be involved. The level for exposure is then determined by comparing the root with the overlying vertebral spine. Usually the tumor is found at this point, but often it is one or more centimeters higher than the level explored. The reason for this is not well understood, but it is probable that the prone position of the patient as during operation, with the head in forward flexion, draws the whole cord up the spinal canal and distorts the relation of the various roots to the overlying vertebral spines. A chart of the relations with the subject in the operative position has not yet been accurately constructed, although the one by Déjerine is the most accurate. A fairly long exposure is therefore advisable for a careful exploration.

September 16th Doctor Frazier performed a laminectomy of the fifth, sixth, seventh cervical, and the first and second thoracic vertebræ.

The accuracy of this procedure was later determined by X-ray examination. The dura was opened, and a normal cord was exposed. Exploration of the cord from the sixth cervical to the fourth thoracic segments disclosed no sign of tumor. The wound was closed and a transfusion given.

The patient's recovery from the anæsthesia was good, but on regaining consciousness he complained of very severe pain in the right arm, overshadowing that on the left side, which, however, was unabated. The right arm was swollen, hot and dry. The least touch was extremely painful, and the hand had to be supported upon cotton in order to give any degree of comfort. The pain was of a burning throbbing nature, somewhat similar to the pain which he had in the left arm, but more severe. There was a vigorous pulsation in the finger-tips, and a capillary pulse was very evident under the finger-nails. The vasodilatation was extreme.

The wound healed well, and the pain in the right arm and hand gradually subsided, with drying and desquamation of the skin. There was very little power in the right hand for several weeks, however. These symptoms were due to the manipulation of the spinal cord and its roots during the search for the tumor, the vasomotor phenomena being rather more pronounced than usual.

At one time it was thought that the right palpebral fissure had approached the left in narrowness, and that the pupils were again equal, but the asymmetry appeared again as the vasomotor phenomena in the right arm subsided.

Two weeks after operation the patient was sent to the X-ray department for examination of the spine to determine the location of the laminectomy. The picture could not be taken before the operation because of the recent previous exposure to X-rays, and the plate obtained in New York had seemed sufficient. The photograph taken at this time is reproduced, Fig. 3. It shows the laminæ of the fifth, sixth, seventh cervical, first and second thoracic removed, just as was contemplated at the time of operation. Moreover there is a fracture of the body of the second cervical vertebra, the causation of which is laid to some chiropractic manipulations a short time before admission to the hospital. The most important finding, however, is a spherical mass the size of a peach occupying the apex of the plural cavity and eroding the vertebral ends of the left second and third ribs. A

FIG. 1.

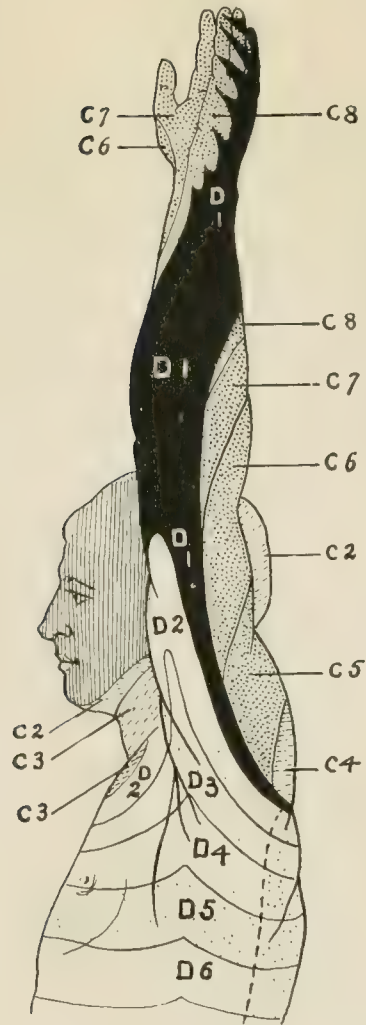


Chart showing distribution of spinal sensory segments on under side of arm. (From Dejerine's *Semiologie*.)

(a)

FIG. 2.

(b)



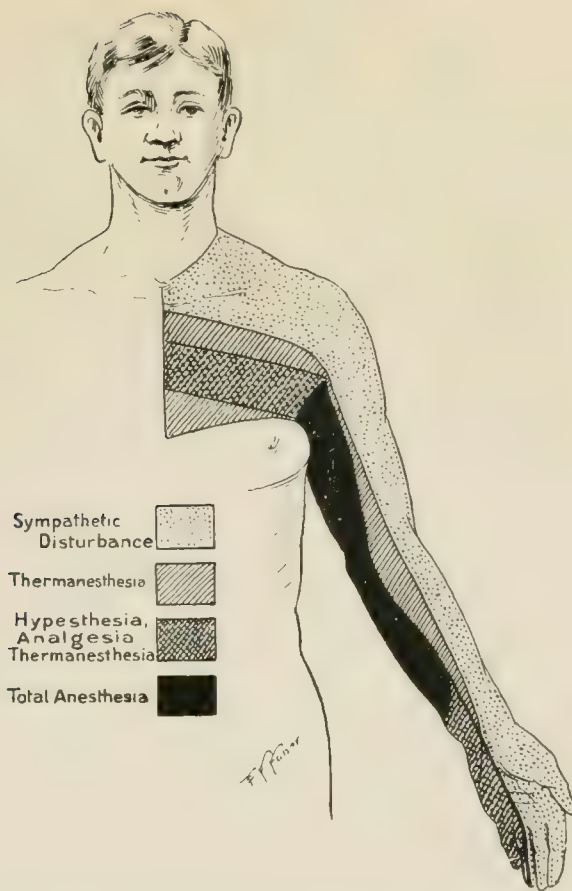
(a) Sensory changes before operation. Shaded area—anæsthesia; clear area—analgesia. (b) Same—dorsal view.

FIG. 3.



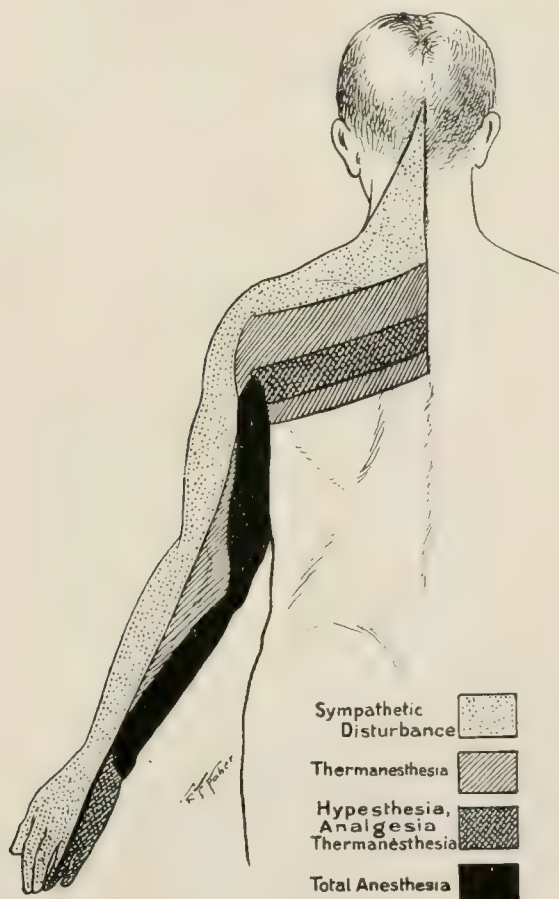
X-ray photograph of spine after operation. Note absence of laminæ and also disappearance of vertebral ends of 2nd and 3rd ribs on left side.

FIG. 4.



Sensory disturbances found on examination after operation (Oct. 6, 1921). The entire shaded area represents sympathetic disturbance.

FIG. 5.



Sensory disturbances found on examination after operation (Oct. 6, 1921). The entire shaded area indicates sympathetic disturbance.

diagnosis of tumor, probably sarcoma, was made. The appearance was so obvious that the plates made in New York were again submitted to Doctor Pancoast, but even in the light of later knowledge he could see nothing abnormal in them. The tumor had become evident in the course of a month.

The further course of the case is what could have been expected. The pains in the right arm having subsided, those in the left have increased both in extent and intensity. The anæsthesia has now extended to the wrist, as shown in the diagram (Figs. 4 and 5), the hypæsthesia has extended beyond this to the finger-tips on the ulnar side of the hand, and in a band across the thorax above the nipple. Analgesia has extended also over the thorax in the second, third and fourth thoracic root distribution, and into the ulnar side of the hand. The atrophy of the small muscles of the hand has become more evident, and fibrillary twitchings are exaggerated. The biceps reflex remains active, but the triceps tendon reflex is absent, and the muscle is undergoing atrophy. In other parts of the body there has been no change. The patellar and Achilles reflexes remain practically as before, and there are no other symptoms of cord compression.

The prognosis of the case of course is hopeless. What can be done in regard to treatment is still undecided. The patient is willing to undergo further operative procedures in order to be relieved of the pain, and it is probable that something will be done, either the tying off of several of the sensory roots, or a section of the antero-lateral column of the cord on the right side in order to cut off the pain and temperature fibres on their way to the brain. Meanwhile the patient must be kept under the influence of opiates to make the pain bearable, although he is averse to the thought of becoming dependent upon them. How death will come must be conjectured. The tumor is located in the midst of a number of important structures, and whether from erosion or pressure they will be destroyed in the course of the growth and expansion of the tumor. The probabilities are that a large vessel will be opened and that the patient will bleed to death into the plural cavity.

In retrospect, with our present knowledge, it is still difficult to see how a correct diagnosis could have been made before operation except with the help of another X-ray examination. This would have been advisable, if merely for the sake of determining the location of the silver nitrate mark before operation, but as far as the discovery for any

mediastinal growth, the plates made in New York but two weeks before seemed to have excluded that possibility, and there was some danger to the skin in re-exposure after so short a time. The neurological symptoms certainly pointed to tumor of the spinal cord, for the patellar reflexes certainly were more prompt than normal, and the left was slightly more prompt than the right. Moreover the slow progression of the symptoms, with the involvement of one root after another pointed to such a lesion. The fact that so many roots were involved is not unusual, for spinal cord tumors are prone to be elongated, lying in the spinal canal and extending over several segments. There are tumors in the neuropathological collection that have reached a length of eight centimeters. It was the appearance of the sympathetic signs however, which pointed so definitely to the presence of a tumor in the immediate vicinity of the cord, and as high as the first thoracic segment, and which determined the performance of the operation.

We were suspicious of two things, first, the absence of more pronounced symptoms of cord compression, that is, spasticity and weakness of the left lower limb; and secondly, the appearance of the mass in the left supraclavicular space which was noted by several physicians, and which later disappeared. Had we had the advantage of finding the mass, we might have judged better regarding its character, but the interpretation of a mass which had lasted but a few days and then had entirely disappeared was impossible. The tumor could have been intravertebral and cause at the present extent of growth only sufficient pressure to produce the exaggeration of the reflexes in the lower limbs. The relatives of the patient were informed before the operation that the tumor might be extravertebral, but they preferred to have the operation and to give the patient the only chance for his life. He has not been made worse by the operation.

NOTE.—Second operation by Dr. George P. Müller exposure of the growth from behind and insertion of radium needles. A small piece of tumor removed and submitted to Dr. Allen J. Smith pronounced to be endothelial carcinoma of pleura.

Pediatrics

EDITED BY JOHN FOOTE, M.D.
Washington, D. C.

THE DIAGNOSIS AND TREATMENT OF POLIOMYELITIS

CLINIC OF A. LEVINSON, M.D.,

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THE diagnosis of poliomyelitis is extremely difficult and requires a consideration of the history of onset, of the course of the disease and of the cerebrospinal fluid findings.

The following two cases illustrate the usual history and symptomatology of acute anterior poliomyelitis.

CASE I.—H. P. This boy is two and a half years old. He has been at the hospital for nearly two months and was sick four days before he was brought to the hospital. He was taken sick with marked constipation and pain in the abdomen. The constipation was relieved by means of an enema. The temperature was 102°F. On the second day the patient was taken to the dispensary. At this time he was able to walk, but complained of weakness in the legs and wished to be carried. He next developed pain all over the body and the fever persisted. On the third day he walked in the morning but could not do it in the afternoon. On the fourth day, when he was brought to the hospital he was unable to move his arms and on the fifth day he could not talk.

This is a typical history—starting with constipation and fever, sometimes accompanied by headache, and with pain and tenderness all over the body. I wish to emphasize that nearly every polio has tenderness at various parts of the body. Fever is also marked. Vomiting may or may not be present. After two to four days of continuous temperature, tenderness and possibly headache, the child becomes paralyzed.

When this patient came here he looked acutely ill; he had a temperature of 102°F., pulse 112, respiration 56. The pupils were equal and reacted to light and accommodation. There was a marked head-drop and slight rigidity of the neck. I wish to point out right here that hardly a case of polio is seen that does not show a head-drop. Sometimes there is associated rigidity of the neck. The lungs were hyperresonant. There was marked limitation of diaphragmatic movement. Breathing was rapid and shallow. There were no râles present.

The abdomen was distended and soft. The liver was just palpable.

The right upper extremity was slightly spastic and had some muscle tone. The left upper extremity was completely paralyzed. Both lower extremities showed flaccid paralysis.

The corneal reflexes were present on both sides. The peroneal reflexes were also present, so was the pharyngeal reflex. The abdominal and cremasteric reflexes, however, were absent. The Babinski, Kernig and Brudzinski signs were negative.

On spinal puncture a clear fluid was obtained under increased pressure. The Nonne and Ross-Jones globulin tests showed slight turbidity. There were 67 cells to the cubic millimeter, consisting of lymphocytes. The Lange and Wassermann reactions were both negative. The white count of the blood was 20,800.

The child to-day, about eight weeks after the onset, has a slight head-drop, not nearly as marked as when he came in. The muscles of the neck have regained some of their tone. He still has muscle tenderness over the upper and lower extremities, especially over the lower. The reflexes are absent and there is no motion in the lower extremities at all. There is a foot-drop on the left side. Some motion is present in the left upper extremity but none in the right upper. The child feels comfortable.

The most important points in this case are that the disease started with constipation, fever and pain in the extremities, and was followed by paralysis on the third day. Muscle tenderness remained all through the disease. Some motion is being regained, but not enough to use the limbs. Foot-drop is developing.

CASE II.—This boy, Jack McD., is 3 years old. He took sick with headache and pain all over his body and vomited once the first

day. The following day the headache was more severe but he did not vomit any more. He was brought to the hospital on October 4th, after having been sick for three days.

Examination showed a head-drop. The pupils were equal and reacted to light. The chest was negative.

The lower extremities showed the extensors of both ankles to be affected.

The corneal, the pharyngeal and pectoral reflexes were present. The cremasteric reflexes, the knee-jerks and Achilles jerks were absent.

The spinal puncture gave a fluid that was clear and under increased pressure. There were 29 cells, all lymphocytes. The globulin reaction was positive; no organisms were found.

Now, after ten days, the child looks fairly well. There is no marked head-drop, but foot-drop is present on both sides. The patient still has some muscle pain in the lower extremity. The reflexes are still absent. This is a very light case of poliomyelitis.

We have 14 cases in this ward at present. This first one has both upper extremities affected. The child in the second bed has an upper left paralysis, the deltoid, biceps and pectorals being involved. She cannot raise the arm because she has a deltoid paralysis. The third child has both upper and lower extremities involved. The fourth child has involvement of both upper and lower extremities. The fifth has both lower extremities involved. In the sixth case there is involvement of both lower extremities, more pronounced in the left. The seventh and eighth cases have both lower extremities involved. In the ninth case the lower left is affected and in the tenth the upper left extremity. The eleventh child has involvement of both lowers and in the twelfth case both lowers and the left upper are paralyzed. The thirteenth has just a facial and the fourteenth a lower left paralysis.

It can readily be seen that most of these patients have paralysis of the lower extremities. Whatever else they have they nearly all have muscular pain and head-drop and nearly all of them have some paralysis or paresis of the abdominal wall. Occasionally this cannot be made out, but it is nearly always present.

Diagnosis.—The most important diagnostic features of poliomyelitis are the acute onset with headache, sore throat and vomiting occasionally, abdominal pain, tenderness all over the body, head-drop, flaccid paralysis of one type or another and cerebrospinal fluid changes.

In the first few hours of the disease one has to differentiate poliomyelitis from grippal infection. A good many cases present in the early stages a typical picture of grippe and the very best physician may not be able to tell whether it is grippe or poliomyelitis. All one can do is to suspect poliomyelitis and watch the case. Very early one of the most important differential points is the cerebrospinal fluid. In the ordinary grippal infection there is no change in the fluid, except possibly an increase in amount and pressure, while in poliomyelitis marked changes are found, such as increased globulin, increased cells and at times some change in the Lange reaction.

On the second or third day one has to differentiate poliomyelitis from meningitis, the differential point being that in a meningitis the reflexes are not absent while in poliomyelitis they are either gone entirely or are weakened. In meningitis, there are also positive Kernig, Babinski and Brudzinski signs and rigidity of the neck. In suppurative meningitis, the cerebrospinal fluid is characteristic. It is turbid. It contains many polymorphonuclear leucocytes and one can usually find the organism in direct smear or culture.

As to tuberculous meningitis, the spinal fluid is of great diagnostic aid if tubercle bacilli are found. When no organisms are found the sulphosalicylic mercuric chlorid test described by Tashiro and Levinson may help in the diagnosis. The onset in tuberculous meningitis is not so sudden as in poliomyelitis. I want to point out, however, that tuberculous meningitis may start in with a sudden onset and simulate poliomyelitis. More than once a diagnosis of poliomyelitis was made in cases of tuberculous meningitis.

The differentiation between poliomyelitis and encephalitis often taxes our skill—those of us who have skill. In encephalitis the reflexes are not lost. The localization of the paralysis is usually in one of the muscles of the eyes. It does not show the extensive paralysis of poliomyelitis, nor is the paralysis flaccid in type as it is in poliomyelitis. The spinal fluid in encephalitis often shows increased globulin, but few cells.

The course of the disease helps make the diagnosis.

There is, of course, polioencephalitis, which is included in the famous classification of Wickmann. The Wickmann classification takes in eight different types: The flaccid spinal, the abortive, the meningeal, the encephalitic, the bulbar, the ascending, the ataxic and

the neuritic poliomyelitis. This classification, however, is not very important. I think one can classify poliomyelitis into three main classes: One, the flaccid, another the bulbar and, third, the polioencephalitis. Of course, there is always an abortive type of poliomyelitis the same as in every other disease.

In the bulbar type of poliomyelitis one may have to differentiate the disease from laryngeal diphtheria. In the last three months I have been called twice to cases of poliomyelitis in which the attending physician made a diagnosis of laryngeal diphtheria. The child could not breathe and as the disease started with a sore throat it seemed to the physician on the case that it was due to diphtheria. In one instance I was asked to intubate. One must remember that in bulbar poliomyelitis there is usually a paralysis of the chest muscles, while in laryngeal diphtheria there is an epigastric retraction. In other words, the muscles of the chest are not paralyzed but are more active than normal because of the obstruction in the larynx. The difference is very important, because if it is laryngeal diphtheria, the patient may be relieved by intubation or tracheotomy, while if it is a bulbar poliomyelitis there is nothing to do, except possibly to administer a little atropin.

After paralysis has set in one may have to differentiate poliomyelitis from post-diphtheritic paralysis. The history of the case and the rapid improvement in post-diphtheritic paralysis may help to establish the diagnosis.

There is one patient on our service now in whom the spinal fluid gave a positive Wassermann test and a paretic Lange curve, but who otherwise presents all symptoms and signs of poliomyelitis. It was only the acute onset of the disease and the flaccid type of paralysis that enabled us to establish the diagnosis of poliomyelitis as over juvenile paresis.

Treatment — There are two types of treatment in vogue, which are wrong. One is no treatment at all, namely to leave the patient alone altogether, and the other is over-treatment. At the very beginning poliomyelitis should be treated the same way as any other acute disease. Give the patient a physic and keep him in bed. When the paralysis develops the most important part in the treatment is to keep the child at rest. It is important in every acute infectious disease to keep the child at rest, but it is much more important during the

early stages of poliomyelitis, because that is the period when nature does her most destructive work and the more rest you give the body, the less destruction will be produced in the cord or in the brain. In the bulbar type of the disease atropin in doses of 1-150 for a child of two years may do some good.

The bed clothes should not be permitted to touch the patient during the very acute stage. There should be some sort of frame over which to place the bed clothes. The child is too sick to have the weight of a lot of bed clothes over him.

Serum of one kind or another has been advocated by many people. In this country Rosenow and Nuzum produced a poliomyelitis serum. Both of these investigators have worked on the assumption that a micrococcus causes this disease. There is a good deal of discussion in the literature as to whether the serum does any good. My experience with the serum has not been extensive enough to warrant an opinion in the matter.

Netter, of France, has advocated convalescent serum from patients who have recently recovered from poliomyelitis. That seems to be a rather rational treatment. At least, I do not believe it does any harm. However, if any serum is to be given, it should be done early before nature has completed the destruction of the affected part because later it surely does not do any good. Of course, a Wassermann test should always be made on the donor.

Hexamethylenamin (urotropin) has been suggested for the early stages of poliomyelitis. Some years ago a certain investigator found that when this drug is administered by mouth it can easily be recovered in the cerebrospinal fluid, and because of that he suggested that urotropin in large doses be given to all cases of poliomyelitis. All I can say for that is, that I do not believe it does any harm. I do not believe, however, that the production of formalin compounds in the cerebrospinal fluid will check the inflammatory process.

As to diet, these children should be given a soft diet. We have changed in a general way the conception of feeding in all infectious diseases. Years ago every infectious disease was treated by milk and water, and since many patients rebelled against milk and vomited it after a short time they received only water. One still finds patients who have been kept on water for ten or twelve days, but this is nothing less than criminal neglect. The metabolism

of the body during the disease is accelerated, and unless some food is supplied the organism cannot fight the disease, and because of that I think every acute infectious disease should have a soft diet. In poliomyelitis it is even more important to feed the patient, for it is not a matter of a few days, but of weeks and months. The food should be balanced but enough should be given to enable the body to pick up some resistance.

Many patients afflicted with poliomyelitis cannot feed themselves because of the paralysis of the upper limbs and because of the head-drop. It is therefore imperative that each patient be fed or at least supervised during the feeding period by a nurse.

A question upon which there is a good deal of difference of opinion is when to start treatment of the affected limbs. There are some who advocate massage and electricity during the first few days after the onset. Experience has taught me that this is wrong. As long as there is muscle pain I believe nothing should be done in the way of active treatment of the paralyzed extremities, and most of these children have muscle pain for three or four weeks. I am surprised to find orthopedic surgeons who insist upon massage during the first few days. The feet, however, should be supported by a box or sand bag so as to prevent foot-drop. If any evidence of deformity is noticed early, orthopedic appliances should be used and of those a cast is the best appliance. A cast put on unintelligently will do much more harm than good, however. For instance, to prevent foot-drop the foot should be placed in an inward and forward position at a right angle to the shaft of the tibia, because the tendency of nature is to produce deformity in the direction of least resistance, namely, downward and outward. In most cases it is advisable not to put on a full cast but just a half cast, or a splint, so that it can be taken off and replaced again.

Do not let the mother and father, or the grandmother insist on your putting on an appliance so that the child can walk. The first three or four months of poliomyelitis needs no brace. The child should be kept in bed for three or four months. That is one of the most important points in the treatment, and one of the most difficult to carry out. I have a case under observation now where on the second day of the disease the mother insisted that I put on a brace so that the child could walk. She could not understand the need of rest. The psychic treatment of the mother, the grandmother and sometimes of

the father is just as important as the treatment of the affected limbs. They must be handled delicately, and tactfully, but you must impress upon them that it will be a long time before the child can get well, and that under the best of treatment the child may have some deformity. However, in order that the deformity may be as slight as possible, the child must be kept in bed with very little treatment in the beginning, and after the muscular pain has subsided one can put on some type of cast, with the child still in bed.

There is one part of the body that practically always needs support, and that is the abdominal wall. In the literature very little attention has been paid to this point except by one or two authors. Upon attempting to sit up, a child with poliomyelitis just crumples down. A good deal of this is due to weakness of the abdominal muscles. Abdominal support is of great assistance in preventing deformity and lagging of the abdominal muscles.

After the muscle pain has gone, muscle training should be started, and that is where a knowledge of anatomy is very important. The physician treating the case should pick out the muscles involved. As a general rule, of course, certain groups are involved. The extensors and gastrocnemius are usually involved in the lower extremities. In the upper extremities the deltoid and biceps are usually involved, and sometimes the pectoralis. These muscles should be first trained by passive massage once or twice a week. Outside of that the child should be left alone. Later the patient should be taught to exercise the affected muscle himself. One who massages poliomyelitis cases must know as much anatomy, or even more, than the physician. It is a question of picking out the affected muscles and treating them. Not every masseur or masseuse is fitted to treat a poliomyelitis patient. It requires a great deal of knowledge and still more skill to benefit any of these cases by massage. All the time, however, the child should be watched for deformities, and as soon as a deformity occurs it should be corrected in so far as possible.

One thing is true—were it not for our lack of knowledge of poliomyelitis there would not be so many cripples in the world. A good many cripples have been cases of poliomyelitis that have gone unrecognized and have received no treatment. In other words, the treatment of poliomyelitis should be scientific and should extend over a period of months and years—if one can retain the confidence

of the family that long, for the patients usually wander from one place to another seeking advice.

No case of poliomyelitis ever recovers entirely. There is always something pathological left, unless the paralysis is so slight that it requires very little treatment. However, if intelligent treatment is given I believe that most cases of poliomyelitis of the extremities will regain at least some function.

TREATMENT OF RICKETS BY ULTRA-VIOLET LIGHT

By DR. LUDWIG F. MEYER

Chief Physician of the Orphan Home and Infants Asylum of the City of Berlin

RICKETS is a folk disease of the present age. In the large cities one rarely sees children between the ages of six months and one year without some rachitic stigma. At the present time every infant in Vienna is suffering from the disease and according to my own experience the number of rachitic children is not less in Berlin. In consequence of the undernutrition during the war the severe forms of rickets have actually doubled in Germany and Austria as compared with the pre-war period. The social-hygienic importance of rickets for children (one need consider only the severe course of bronchitis, pneumonia and measles) and for adults (permanent deformity) has kept alive the interest of physicians of all countries in the treatment of the disease, ever since the first description of its clinical picture 300 years ago.

Hitherto the treatment of rickets was not based on etiological factors, for despite all investigation the cause of the disease is still absolutely undiscovered. Every factor that impairs the physical vigor as a whole has been held responsible for the development of the disease. Heredity, artificial feeding, nutritional disturbances, deficiency of vitamins in the diet, infections, disorders of the internal secretions, and (not last) the domestication incident to our cultural development have been taken into account in considering the causative agents. For each of these factors certain proofs have been advanced, but none has been generally accepted. Perhaps a group of these factors are combined in the production of rickets.

The nature of the pathological processes is just as undefined as the cause. Although many physicians consider rickets as a local disease of the bones, we are reverting more and more to the standpoint of Glisson, its discoverer, that the disease originates in a general disorder of the whole organism, in a dyscrasia. In the light of such a concept the anatomic changes in the bones are not considered as primary factors but as the secondary result of an altered metabolism. Accord-

ing to Schmorl the pathological process is based in the final analysis on the fact that there is an interference with the deposition of calcium salts in those skeletal parts which absorb them during the normal period of growth. The basis for this failure of calcification cannot lie in primary deficiency of calcium, for experiments on growing animals fed on a calcium-free diet demonstrate anatomic changes of the bones differing absolutely from those of rickets. Moreover the exhibition of calcium salts in large quantities has never cured a case of rickets. There is no calcium deficiency, but a disturbance of the chemical equilibrium in the sense of an inability on the part of the osteoid tissue to fix the available calcium. There is an absence of "calcium salt receptors" (Kalksalzfänger of Pfaundler), which attend to the apposition of calcium in osseous tissue. Here is the key to the biochemical problem of rickets. Calcium salt receptors must be manufactured by the metabolic processes in order to assure the fixation of phosphoric acid and calcium in rachitic bones. Despite our ignorance of the etiology and nature of rickets, empiric practice has advanced beyond theoretic knowledge and has revealed methods of treatment that have yielded unassailable results. The treatment has been directed along three different lines: Dietetic, medical and physical.

The dietetic treatment of rickets aims at an improvement of the calcium balance. It is successful in cases in which friable, hard soap stools indicate an excessive excretion of calcium by way of the intestines. Whereas this condition was formerly corrected only by decreasing the amount of fat in the feedings, we now know that it may be avoided by an increase of the carbohydrate constituents and by a modification of the relative percentages of carbohydrate and fat in favor of the former. The quality of the carbohydrate (addition of flour and malt extract) is also of great importance in the correction of soap stools.

In conformity with past experience the dietetic treatment includes also the restriction of the total quantity of milk and the substitution of fruits, vegetables, potatoes and meat at an earlier age and in greater quantity than is normally indicated. A. F. Hess and L. J. Unger¹ have demonstrated by clinical experiment the value of the addition of vegetables in the dietetic management of rickets. In fact, the addi-

¹ HESS, A. F., AND UNGER, L. J.: *J. A. M. A.*, lxx, 900; lxxix, 1583.

tion of vegetables increases the calcium balance to a marked degree, the expressed juices of fresh vegetables being more efficacious than cooked vegetables (Freise and Ruprecht²).

For the last century the remedy for rickets (κατ' ἐξοχήν) has been cod-liver oil, which Bretonneau introduced into the pharmacopeia. Time and again certain modifications of the cod-liver oil have been prescribed, including combinations with phosphorus and calcium. At present the following prescription is in vogue in Germany:

Calcii phosphatis trib. 10.

Olei morrhuae q. s. ad 100.

Sig: Teaspoonful twice a day.

There has never been any doubt as to the effective influence of cod-liver oil on rachitic processes. Cod-liver oil not only produces a favorable result in cases where the disease is already developed but it is also a specific prophylactic. Hess and Unger have demonstrated through tests on negro children that the early prophylactic administration of cod-liver oil protected practically every child from rickets, and that the period of treatment and the amount of cod-liver oil bore a direct relation to the percentage of successes. Metabolism studies have fully confirmed clinical experience on this point. Birk, Schabad and Schloss have made accurate determinations of the favorable influence of cod-liver oil on calcium retention in rachitic children.

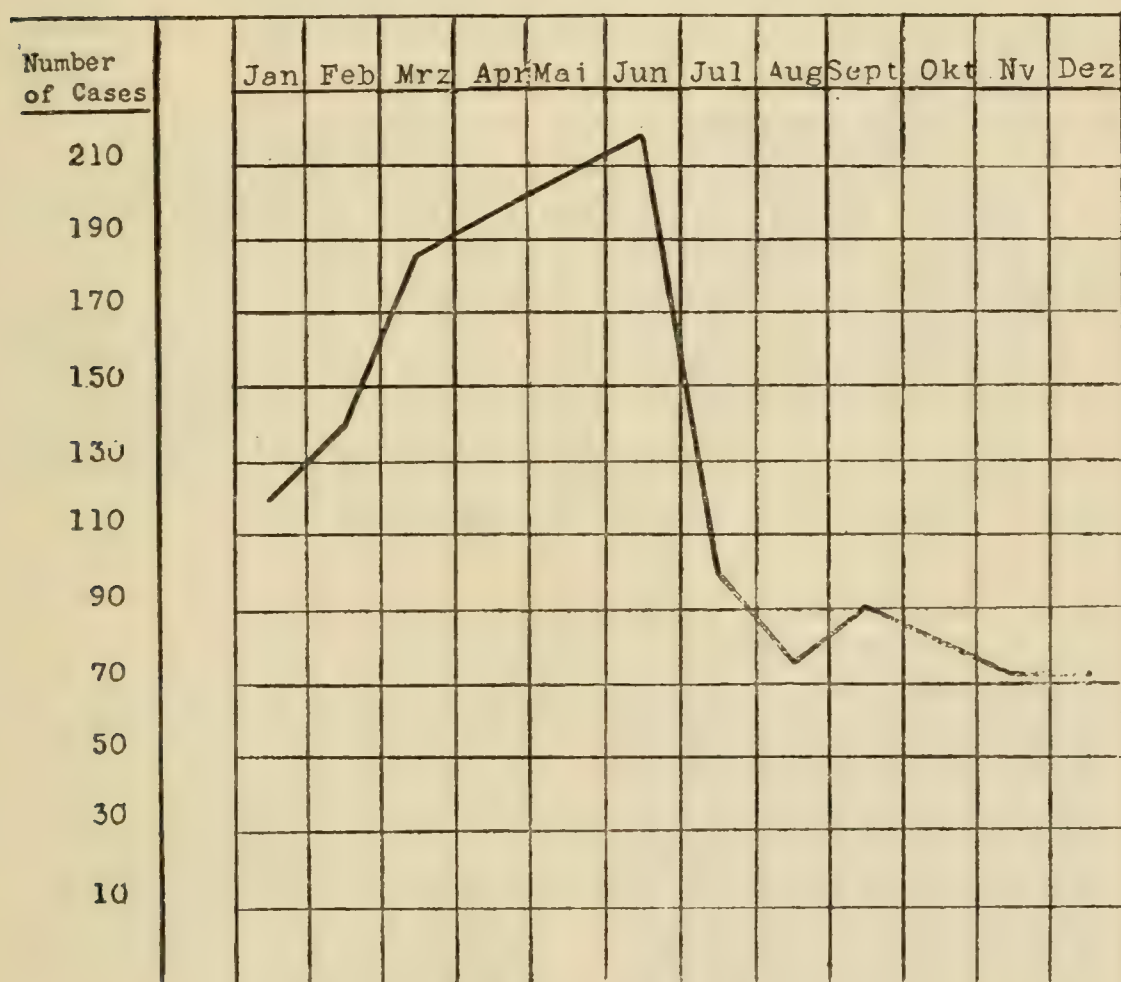
The dietetic and medical problem of rickets received additional elucidation by the consideration of the disease as an avitaminosis, a theory which is strongly supported by American and British investigators. Of the three groups of accessory food factors, the fat soluble A, found in whole milk, butter and cod-liver oil, is of particular interest here, for a deficiency of this vitamin is made responsible for the development of rickets, which is held to be curable by its administration. It is certainly true that the antirachitic principle is not contained in the fat soluble A factor alone. This is learned from our experience with the favorable effect of fat free vegetable feedings on rickets and calcium metabolism, to which reference has already been made, and from the following observation by Hess and Unger:

A diet very rich in antirachitic factors (whole milk, cream and butter) did not prevent or cure rickets, whereas a diet that contained little of the fat soluble vitamin but was rich in water soluble factors

² FREISE and RUPRECHT: *Jahrb. f. Kinderheilk*, xix, No.2.

B and C (skimmed milk, yeast, flour, sugar, orange juice, etc.) did not cause a greater number of cases of the disease. Those who maintain the vitamin theory, a theory which is very plausible when applied to the problem of rickets, must first acknowledge that the

FIG. 1.



Frequency of rickets during the various months of the year.

antirachitic principle is found in the fat soluble as well as in the water soluble factors of the diet.

The advance of knowledge, which was marked in the first place by the vitamin investigations, has forced other tried methods of treatment into the background, and unjustly, too. For even the ancients, who were excellent observers, have always emphasized the therapeutic influence of light, air and sunshine. It is probably not mere coincidence that the morbidity rate of rickets, as was first demonstrated by

Kassowitz, increases markedly at the end of the winter and in the early spring, and decreases rapidly in the summer.

The accompanying chart (Fig. 1) by Fischl illustrates clearly the rise of the curve during the winter and spring.

It has also been known for some time (H. Neumann³) that rachitic affections are rare in sunny mountainous regions. To be sure it was still a question as to what factor to attribute the favorable influence of the sun and mountain climate, whether it was due to increased isolation, to lower barometric pressure, or to the avoidance of domestication, which many have considered injurious.

These questions can be answered now that we have artificial rays (since 1895). Qualitatively the components of sunlight may be differentiated as (1) heat rays, (2) light rays, and (3) ultra-violet, chemically active rays.

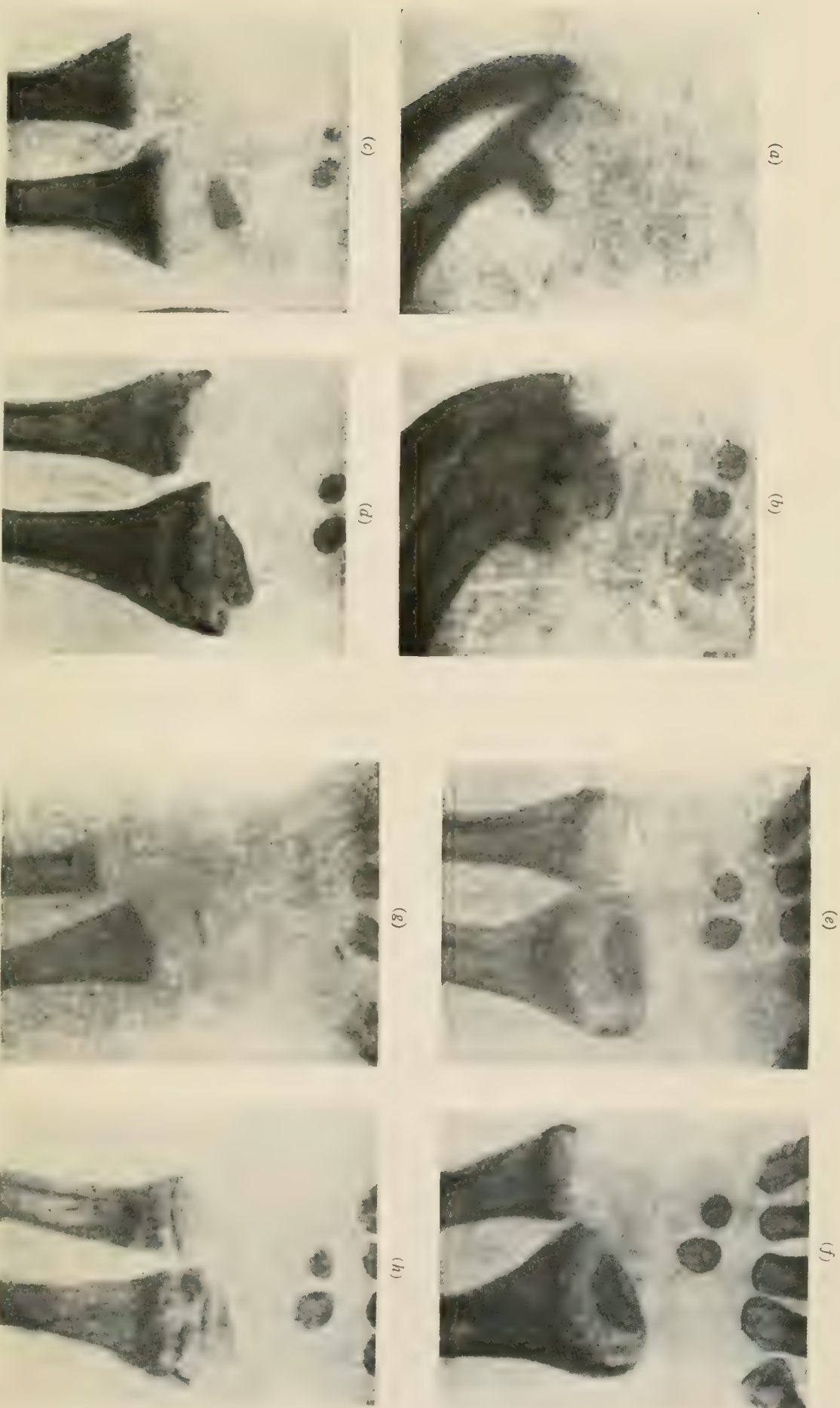
As soon as recent technical advances placed at our disposal the chemically active rays beyond the violet border of the spectrum, rays which are known to be present in the sunlight of mountains and seaside, it was readily demonstrated that neither the heat rays with long waves nor the visible light rays had any influence on the etiology, course and prevention of rickets. Huldshinsky⁴ was the first to institute systematic attempts at the treatment of rickets with artificial ultra-violet light (the mercury quartz lamp according to the Hanau system).

The technique of irradiation is very simple. Treatment is given three times a week, the anterior and posterior surfaces of the body being alternately exposed to the rays, consideration being given to protecting the eyes. The distance between the lamp and the child should at first be maintained at 100 cm., gradually this distance may be reduced to 60 cm. The first sitting should be for three minutes, and each sitting may be increased by from one to three minutes until the maximum of from twenty to thirty minutes is reached. After an apparent result is obtained, usually after a course of treatment covering about four weeks, it is advisable to omit irradiations for from eight to fourteen days, and in severe bed-ridden cases to begin a new series of sittings. As a result of the exposure, an erythema usually

³ NEUMANN, H.: *Deutsche med. Wchnschr.*, 1909, No. 9.

⁴ HULDSCHINSKY, K.: *Die Behandlung der Rachitis durch Ultra violettbestrahlung*, *Ztschr. f. Orthop. chir.*, xxxix, 426; *Strahlentherapie*, xi, 435, and *Putzig Therap.*, 1920, No. 8.

FIG. 2.



Radiographs illustrating the effects of ultra-violet light on bone calcification in rickets. (a) G. L., 3-year old girl, before treatment by irradiation, and (b) after two months of treatment. (c) R. G., 2 $\frac{1}{4}$ -year old boy, before treatment, and (d) after one month of treatment. (e) I. B., 2-year old girl, before treatment, and (f) after three months' treatment with phosphorous and cod-liver oil. (g) E. L., 4 year old girl, before treatment, and (h) after one month of treatment by irradiation. (From Strahlentherapie, XI.)

develops, gradually resulting in increased pigmentation of the skin. If the distance between the lamp and the body is not sufficient or if the patient has cutaneous hypersusceptibility a harmless burn of the skin may be produced. It is always advisable, therefore, to reduce the distance very carefully. Dietetic treatment is unnecessary during the phototherapeutic course. On the hypothesis that the diseased bone required from 30 to 60 grams of calcium, Huldchinsky at first prescribed calcium phosphate, 1-1.5 gram daily. If the diet was rich in calcium this prescription proved superfluous. It is advisable to prescribe calcium only in those cases in which the diet is very low in calcium, *e. g.*, breast milk or butter and flour gruels.

Huldchinsky summarized the results of ultra-violet irradiation in the following sentences:

1. The therapeutic effect of quartz light irradiation is invariably apparent in all forms of rickets.

2. Recovery results more promptly than with any of the methods employed heretofore.

3. The effects of irradiation are lasting and the regenerative process is active for at least two months after interruption of treatment.

4. The duration of treatment embraces (a) from two to four weeks in infants, (b) from one to two months in children aged one year, (c) from two to six months in children aged two to four years, and (d) up to nine months in older children.

Huldchinsky demonstrated röntgenographically that calcification of the bone paralleled the clinical improvement. This fact is best illustrated in his original röntgenograms. (See Fig. 2.)

Following the first publication of the treatment of rickets with the quartz light, considerable skepticism prevailed in scientific circles regarding the value of the method. Until that time the indications for the use of ultra-violet light were much too vague and there were too many uncritical reports on the therapeutic results of artificial sunlight in the most variegated diseases; tuberculosis, anemia, skin diseases, etc. Like most of my colleagues I did not at first place much hope in the success of the quartz lamp. In this case, however, my skepticism proved to be unwarranted. After one year's experience with phototherapy in rickets I may say that it has proved an excellent measure and that it has done everything that Huldchinsky

promised for it. It is equal, perhaps even superior, to the medical and dietetic treatment, and possesses the advantage of being applicable to those cases in which the administration of cod-liver oil and vegetables is impossible, in tropholabile infants, in premature, debilitated and undernourished children, and in children with a tendency to gastrointestinal disorders. Ultra-violet light may be designated to-day as a

FIG. 3.

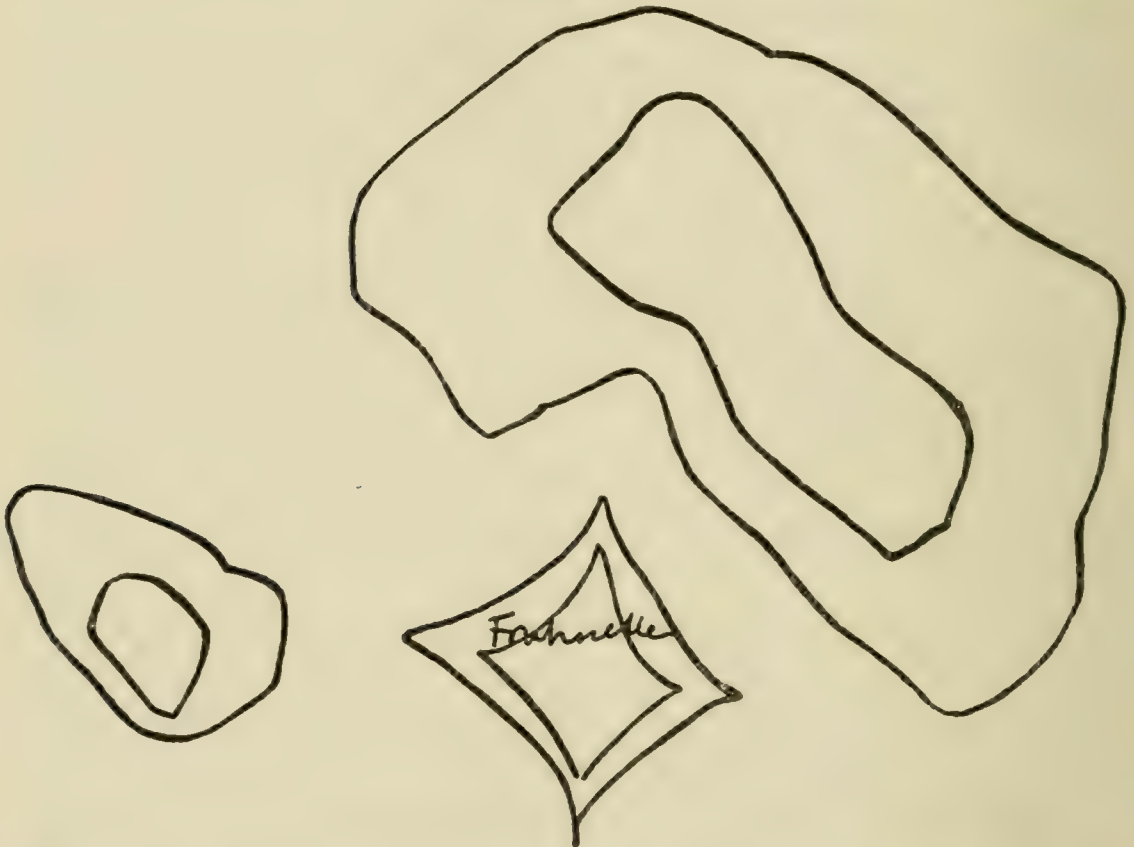


Fig. 3. Influence of irradiation on craniotabes in case of G. A., aged 3 months, after eight treatments in eighteen days. The outer line indicates the areas of softening at the beginning of treatment (Feb. 1), the inner line the findings at the last sitting (Feb. 18). Craniotabes healed, March 7.

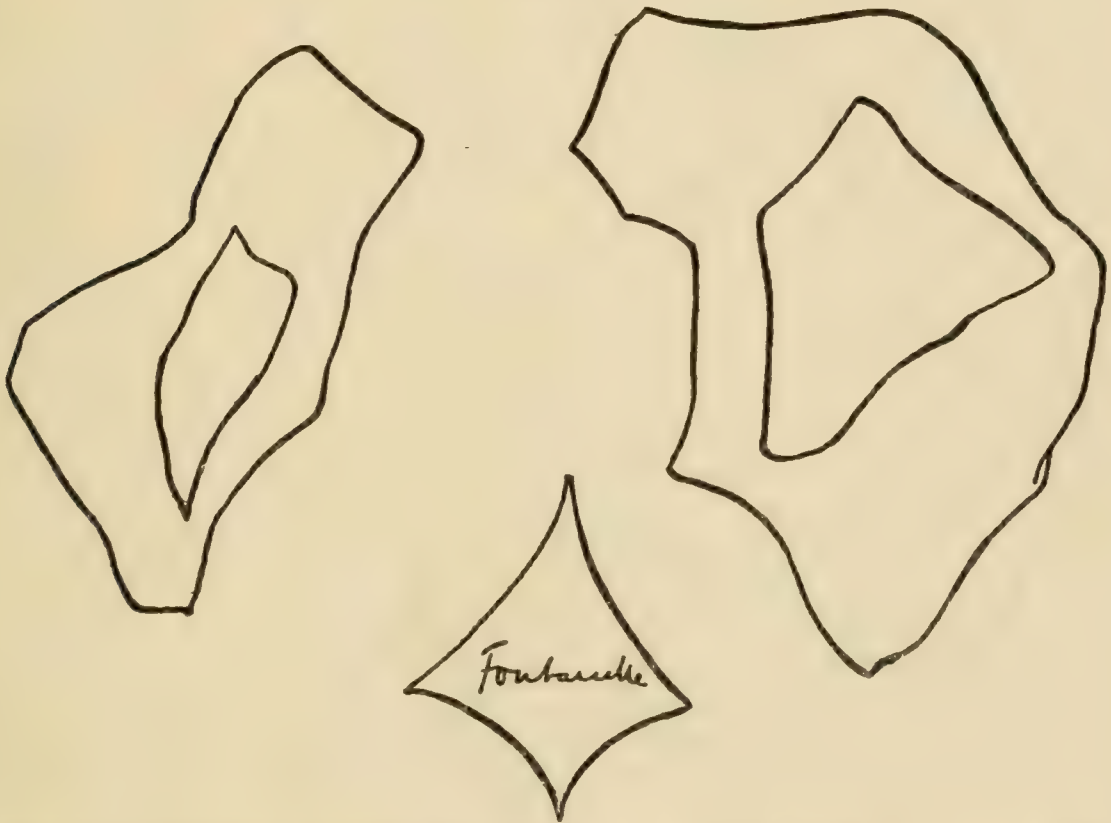
specific treatment for rickets, a method that for practical and theoretic reasons is deserving of the interest of physicians.

I have already gained practical experience with a considerable number of rachitic children, and I have very rarely encountered one who was refractory to the treatment. As I do not desire to fatigue my readers with reports of case histories, I have selected only one clinically demonstrable and comprehensible symptom of rickets as an index of the progress of recovery and I have therefore recorded the behavior of craniotabes under treatment. (See Figs. 3-6.)

A record of the area of softened bone was made as follows:

A closely fitting cap was made of thin linen, and drawn over the occiput. Through this the softened areas were palpated and their outlines were traced on the cap by means of a lead pencil. The extent of these areas is indicated in the figures reproduced herewith. Before treatment the patients had been under observation for varying periods, but had shown no tendency to improvement of the craniotabes. In Case 4 (Erwin M.) the condition had been unsuccessfully treated

FIG. 4.



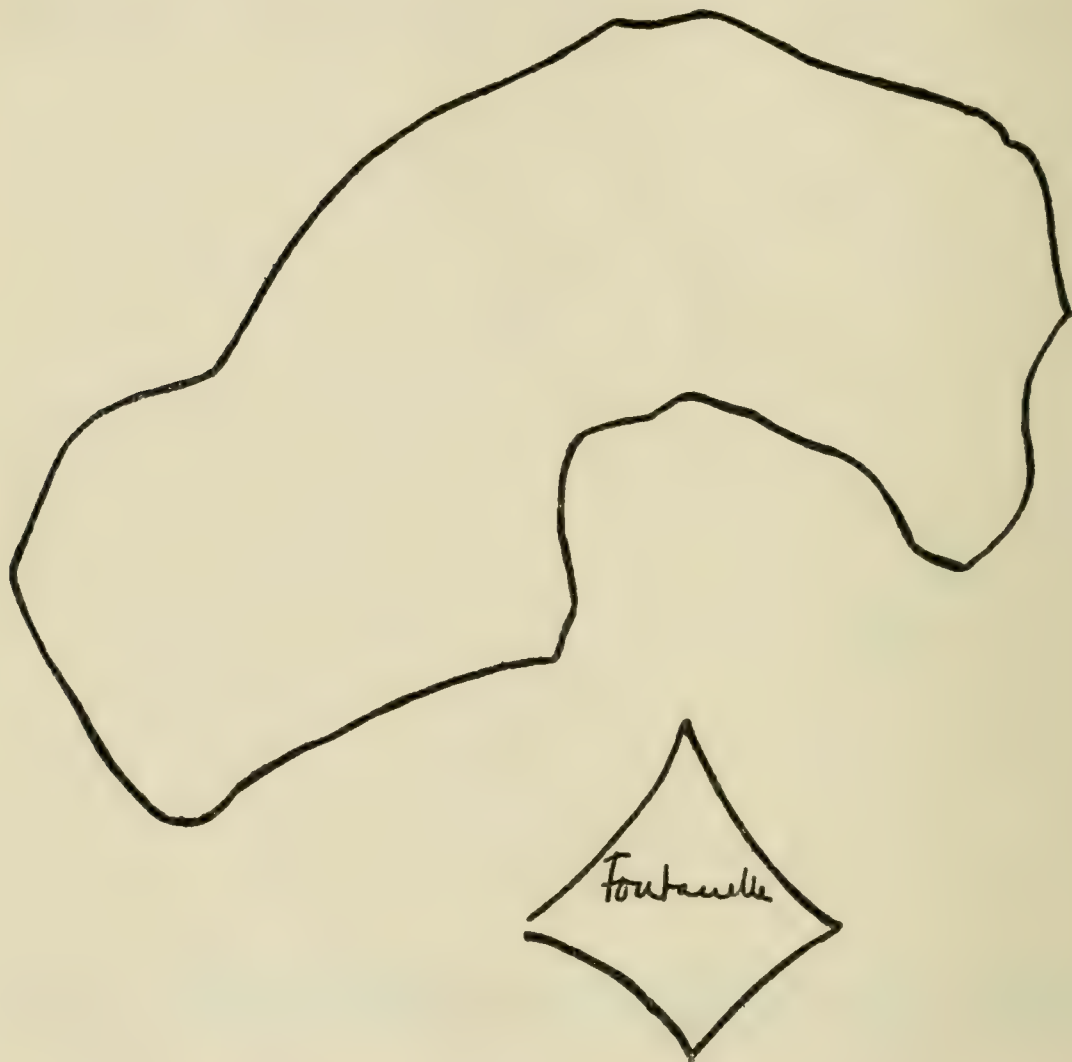
Effect of nine treatments in seventeen days in case of M. K., aged 10 months. The outer line indicates area of softening on Jan. 23, 1920, the inner line on Feb. 8. Craniotabes healed on Feb. 20.

with cod-liver oil for almost five weeks. After a few sittings, three weeks after the beginning of treatment, there was a decided reduction of the areas of cranial softening and after four or five weeks the craniotabes was cured (in Case 4 only a small area remained.)

The four cases referred to show the typical course of healing observed in numerous instances. The most notable feature is the promptness with which the curative reaction sets in. As a general rule consolidation of the softened areas of the cranium and a closing of the fontanelle may be observed after three or four treatments. Calcification proceeds even more rapidly, therefore, than with the

cod-liver oil treatment (Rosenstern⁵). Objection might be raised on the ground that craniotabes often heals in the course of several weeks without treatment. Against this I may state that, as has been mentioned before, the treated cases were subjected to preliminary observa-

FIG. 5.



Effect of eleven treatments in twenty-five days in case of E. K., aged 6 months. The craniotabes was completely healed five weeks after beginning of the course.

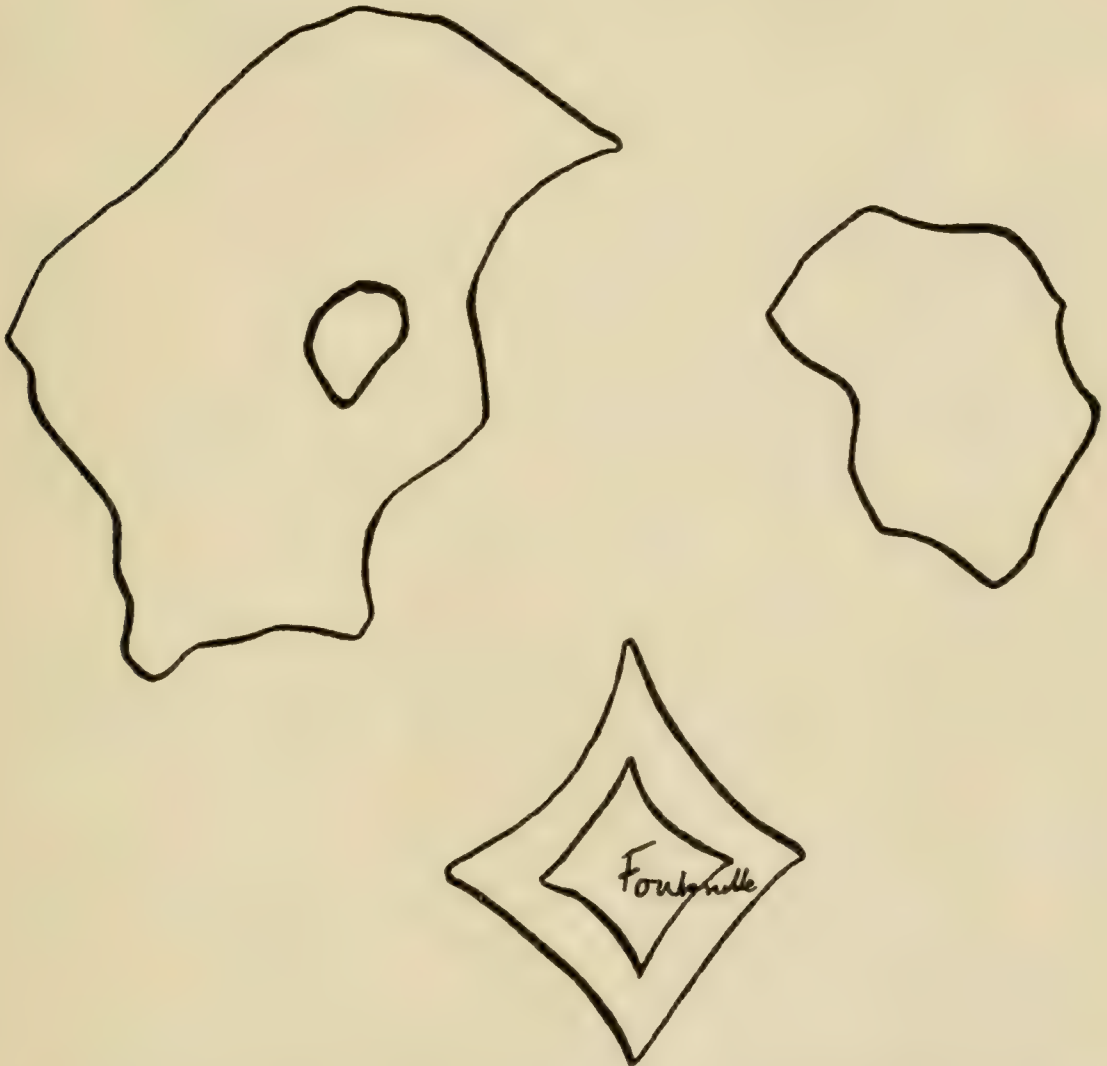
tion, that other methods of treatment had no influence on the craniotabes and that, finally, improvement followed promptly after the first few exposures to the rays.

It cannot be claimed that the influence of phototherapy is equally evident on the other symptoms of rickets. If the craniotabes was treated at the first sign of rickets, the children were spared the well-

⁵ ROSENSTERN: Zur Wirkung des Lebertrans auf Rachitis und Spasmo-phile Diathese, *Berlin klin. Wchnschr.*, 1910, No. 18.

known late manifestations of the disease, the rosary, epiphyseal thickening, etc. I wish to emphasize the improvement in static function as especially striking. Children at the end of their first year who were unable to make any use of their limbs learned to stand after

FIG. 6.



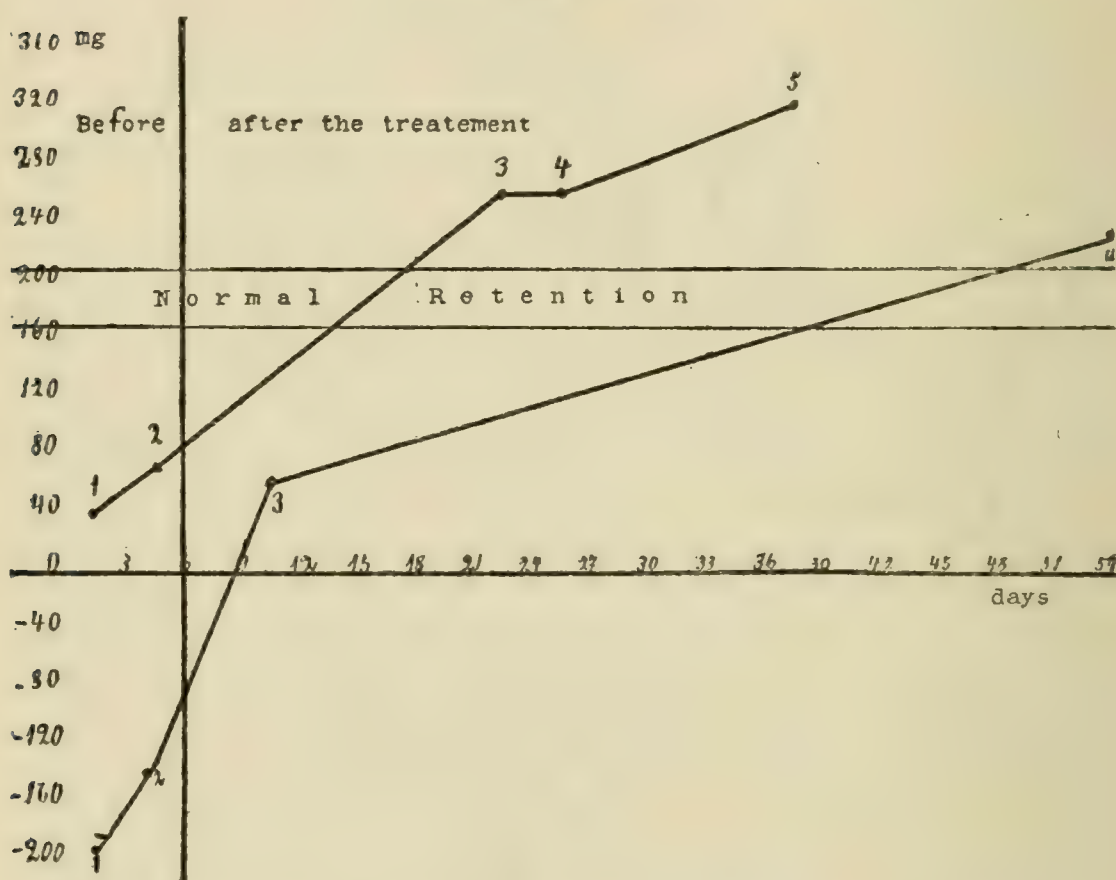
Effect of eleven treatments in thirty-five days in case of E. M., aged 11 months, who had been treated for five weeks with cod-liver oil without result. The outer lines indicate the softened areas before treatment, the inner lines the findings five weeks later.

the first few treatments. The laxity of the muscles, which Bing considers a specific characteristic of rachitis on the basis of anatomic findings, showed definite improvement. In many cases the meteorism decreased. The sweats and the tendency to furunculosis were so favorably controlled. Fractures seemed to consolidate more rapidly than under cod-liver oil treatment, and the susceptibility to fractures and infraction seemed to be more quickly obviated. We did not

obtain any demonstrable effect on existing changes of the blood, and this experience coincides with observations on anemia in infants and children. No success was recorded in cases of osteoporosis or osteopsathyrosis. Finally, it cannot be expected, of course, that existing deformities of the extremities are corrected by phototherapy.

Treatment for four weeks usually sufficed for infants, in older children more time was required to effect a cure. On the whole,

FIG. 7.



Calcium oxide balance as determined by analyses during several stages of treatment.

I can confirm the rule laid down by Huldchinsky that the older the child, the more protracted the recovery. The number of years of age corresponds approximately to the number of months of treatment.

Ultra-violet light has proved effective not only in the treatment but also in the prophylaxis of rickets, a factor which seems of greater importance from the point of view of public health. It is a matter of common experience that all premature infants as well as twins—namely all children born before term—become rachitic. Whereas a child born at term shows the first signs of rickets at about six months

of age, a premature or debilitated infant will develop craniotabes as early as the second or fourth month. The early and invariable development of rickets in this group of children was attributed to the insufficient calcium depot with which they are endowed. I doubt if this explanation suffices. As the prophylactic administration of calcium salts never prevents rickets, some special disorder based on inferior organic activity must supplement the deficiency in calcium endowment. To prevent rickets in premature infants I have therefore applied ultra-violet rays at the first sign of craniotabes, and even before this warning, systematically from the second month. The success of these prophylactic treatments was striking. Never before have I seen premature infants develop with so few signs of rickets, any existing craniotabes healed rapidly and the rosary and rachitic changes of the extremities did not develop. At the end of their first year the children could stand, and during the first months of the second year they learned to walk.

Herewith I append a brief illustration of this:

R., premature infant, weight at birth 1500 grams, developed craniotabes in the fourth month. Under phototherapy the craniotabes healed perfectly. At seven months the child could sit erect without any support, at ten months it could stand alone. Its general development was excellent.

On the strength of my experiences I feel justified in warmly recommending phototherapy during the second or third month for the prevention of rickets in premature and debilitated infants.

A few words about tetany, the laryngospastic form of which frequently develops on a rachitic basis and which is even to-day considered as a symptom of rickets by many authors. Phototherapy has also been recommended for tetany,⁶ but its success has not been demonstrated so clearly as in rickets, perhaps because under the influence of irradiation the calcifying bone draws on the calcium supply and causes a deficiency of calcium in the blood and tissues. Nevertheless, cases have been reported in which artificial heliotherapy produced a decrease in the number and severity of the attacks of laryngeal spasm and reduced the mechanical and electrical irritability. In addition to successfully treated cases there are, however, also refractory ones. Perhaps it would be useful to supplement the photo-

⁶ SACHS, F.: *Jahrb. d. Kinderheilk.*, 1920, xciii, No. 3.

therapeutic course by the administration of calcium (calcium chloride, 5-6 grams a day), in order to forestall a deficiency of calcium in the blood.

What changes in the metabolism balance occur under the influence of phototherapy? It was of particular interest to determine if the biochemical reactions, in relation to calcium and phosphoric acid metabolism especially, correspond to the clinical improvement in rickets. In order to test this question, Doctor Lasch, my assistant, made studies of the nitrogen, calcium and phosphorus metabolism in young rachitic infants (preferably premature infants about three months of age) with well-developed craniotables, before, during, and after phototherapeutic treatment. No changes were made in the diet of the children during the entire period of the studies, so that the results of the tests can be fairly attributed to the influence of the physiotherapeutic factors. I have selected from the whole series two studies which were made at an especially appropriate stage of the disease.

Study 1. DAILY CALCIUM BALANCE (Three-day Period), EXPRESSED IN MILLIGRAMS

Ca O Retention	Absolute	Per Cent. of Intake
I	45	14.5 (Before irradiation)
II	68	20.5
III	257	55. (After 2-3 weeks of irradiation)
IV	269	57.6
V	320	61.3

*STUDY 2.**

Ca O Retention	Absolute	Per Cent. of Intake
I	-194	(Before irradiation)
II	-141	
III	56	17 (After 5 days of irradiation)
IV	233	65 (After 1 1-2 months of irradiation)

* See also Fig. 7.

In the first study, calcium retention was recorded even before the irradiation, but it was subnormal, the daily quantity being 45 (14.5 per cent.) and 68 (20.5 per cent.) mg., considerably below the norm (170-210 mg.) established by Orgler. After six exposures to the rays within two or three weeks the calcium retention had increased to 257 (55 per cent.), 269 (57.6 per cent.) and 320 (61.3 per cent.) mg., that is, above the normal daily balance.

The second study yielded results particularly favorable to our hypothesis. The analyses were made during the stage of florid rickets with the loss of calcium. In a preliminary period of three days this loss as expressed in CaO represented 194 and 141 mg. Irradiation was instituted, and after a very short time, following three treatments within five days, there was a marked change in the calcium oxide metabolism. On the same diet as during the preliminary test there was now a positive calcium balance of 56 mg. per day. Further treatment during the succeeding six weeks brought this positive calcium balance up to 233 mg. per day. Just as in the preceding study this represented a positive calcium balance in excess of the norm, a metabolic symptom which was indicative of the clinical improvement. It should be stated that the phosphorus balance in general corresponded to the calcium balance in the same order.

The results of these metabolism studies confirm fully the clinical observation of the cure of rickets by ultra-violet light. Under the influence of the ultra-violet rays the subnormal calcium balance, or even calcium loss, was transformed into calcium retention in excess of the norm, and this increased calcium retention lasted beyond the stage when there was evidence of the complete clinical cure of the rachitic process, i.e., the craniotabes. Under the influence of the quartz light this basic change in calcium metabolism set in with extraordinary rapidity. Three exposures to the light sufficed to change a negative calcium balance into a positive balance, though still below the norm. The promptness of this modification of the metabolism may really be interpreted as indicating that the pathologic process in the bones and cartilages does not constitute the essential feature in the clinical picture of rickets, for it is hardly conceivable that anatomic changes can be fundamentally improved in so short a time. The hypothesis of a primary disturbance of the intermediate metabolism is rendered more probable by these findings.

In order to study more fully the mechanism of the physico-chemical reactions, it is of prime importance to determine whether the ultra-violet rays alone are the decisive factor in the production of this therapeutic effect. As the quartz light, though made up principally of ultra-violet rays, also contains other visible rays of greater wave-length, the possibility that these play a part in the antirachitic action of the quartz light cannot be rejected forthwith. To decide

this point, other phototherapeutic measures were applied; these were deficient in ultra-violet rays but contained many visible rays and heat rays. One of these, for example, is the Aureol light, the spectrum of which approximates that of the sunlight on the plains of Continental Europe. This was tried in numerous cases but proved absolutely ineffective. Heat waves were also tried without any success. The definite proof that only the ultra-violet rays of the quartz light have an antirachitic effect is furnished by the effect which followed the interposition of a medium that absorbs the ultra-violet rays but does not effect the other light rays. Such a medium may be found in a very thin glass plate, or the so-called "uveofilm" originally recommended for the prevention of erythema from quartz light in patients with sensitive skin. Just as soon as an ultra-violet absorbent medium was introduced between the quartz light and the patient, the light was devoid of all influence on rickets.

In what manner does ultra-violet light act? The rapidity with which metabolic changes set in after irradiation speaks against a direct action on the diseased bone. This point may even be pushed further and all direct action may be eliminated, as the same successful results set in even if some area of the body far removed from the affected part be exposed to the light. Thus, for example, the craniotabes may be cured by irradiation of the trunk only, the rest of the body being protected from the rays by means of black cloths. Ultra-violet light can only act indirectly, therefore, through the medium of the skin, blood, or even the deep-lying glands of internal secretion. The short ultra-violet rays can hardly penetrate to the deeper layers of the body. Although Gassul⁷ has lately reported marked effects of ultra-violet light on the spleen, liver and kidney, it is generally assumed that absorption occurs in the superficial cutaneous layers (epidermis and papillary layer). Presuming that this assumption is correct, we are forced to abandon the idea that the glands of internal secretion, such as the suprarenal and the parathyroid glands, which regulate calcium metabolism, are stimulated functionally and pour antirachitic principles into the blood. There remains then the idea of a superficial tissue reaction induced by the ultra-violet rays. In the first place, we must then consider a reactive stimulation of the skin as a

⁷ GASSUL: *Strahlentherapie*, ix, No. 1, and x, No. 2.

result of the irradiation, similar to that assumed to occur in the cure of tuberculosis by heliotherapy. The therapeutic effect of sunlight in such cases has been explained by the hypothesis that some kind of internal secretion of the epithelium of the epidermis is stimulated, which by generating and increasing the protective elements promotes the cure of tuberculosis. Block⁸ and Hoffman⁹ attribute special biologic importance to the production of protective and therapeutic elements by the skin (termed esophylaxis), and they believe that in consequence of the esophylactic activity of the skin the vital internal organs are protected from the pathogenic microorganisms, or at least they must combat only a relatively small number or attenuated forms of these.

The esophylactic function of the skin, stimulated by the influence of the ultra-violet rays, may be a factor in the cure of rickets. It is conceivable that hormone substances are formed from the skin through the medium of internal secretion and that these substances, acting as calcium salt receptors, stimulate the fixation of calcium by the rachitic bones and thus initiate the healing process.

In addition to the skin the activity of the blood must also be taken into consideration. The body reacts to irradiation by a considerable hyperemia of the skin, so that of all the tissue elements of the body the circulating blood constitutes the photoacceptor par excellence (Neuberg). The blood might respond in two ways to the radiant energy. It may readily be imagined that the blood or the erythrocytes produce calcium fixing hormones, analogous to the esophylactic function of the skin. Secondly, the irradiation may possibly produce a change in the colloidal composition of the blood, thereby permitting an increased transport of calcium to the bones. It is known that the transport of water insoluble calcium soaps and phosphates is made possible only by the colloidal composition of the blood or its high albumin content (Hofmeister;¹⁰ Orgler.¹¹) If an increase in the colloidal substances is induced by the irradiation, then a greater percentage of calcium salts will be carried by the circulating blood to the bones which are hungry for calcium. To be sure, this hypothesis

⁸ BLOCK: *Cor. Bl. f. Schweizer Aerzte*, 1917, No. 39.

⁹ HOFFMANN: *Deutsche Med. Wchnschr.*, 1919, No. 45.

¹⁰ HOFMEISTER: *Ergebnisse der Physiologie*, 1910, x.

¹¹ ORGLER: *Jahrb. d. Kinderheilk*, lxxxvii, 459.

does not even take for granted that a diminution of the colloidal calcium fixation substances has been demonstrated in rickets.

In view of the multiplicity of hypothesis it seems to me superfluous to discuss the mode of action of the ultra-violet rays; the problem is only of heuristic value. The practical result of phototherapy in rickets, however, stands forth and is of special interest at this period when the treatment of the disease is aimed primarily at nutritional factors based on the vitamin theory.

Possibly the dietetic and physical methods of treatment reach the same end somewhere along the way by acting from different angles toward the elaboration of those unknown calcium salt receptors which aid in the fixation of calcium by the diseased bones and thereby lead to the cure of rickets.

THE TREATMENT OF BIRTH IN CONTRACTED PELVIS

By THE LATE PROFESSOR FRIEDRICH SCHAUTA,

Dictated to me shortly before his death.

English by B. Lewis

It is not so very long ago that the opinion was held by many gynecologists that there was nothing new to be said about obstetrics. How wrong this opinion was, is shown in almost all its branches by the reformatory movements begun some decades ago.

One of the most important of these reformatory movements is in reference to the treatment of contracted pelvis. The reformatory movement has brought about a fundamental change in the treatment of birth with contracted pelvis, and, though by no means completed, its future development may be foreseen.

The operative procedures applied in contracted pelvis may be classified as (1) the indicated cases, (2) the prophylactic cases, (3) the surgical cases.

Class 1 includes the cases which are *indicated*, not so much by the contracted pelvis, as by other birth anomalies, which, however, stand in causal relation to the contraction of the pelvis. Their operative removal is necessary, but would be indicated also without complication in contracted pelvis.

Among these operations I would mention:

- Deep forceps;
- Version in transverse position;
- Low extraction;
- Craniotomy with dead child;
- Decapitation.

Class 2.—Among the prophylactic operations are:

- Artificial interruption of pregnancy;
- Abortion or premature birth and the so-called prophylactic version.

Many count the high forceps among these measures.

Class 3.—The so-called surgical operations are those in which the integrity of the maternal body is injured in favor of the child, whether the space of the pelvic canal is enlarged by dilatation of the pelvis

(symphysiotomy) or birth in the natural way is wholly desisted from, and the child is extracted on an operatively created path, through the abdominal walls and the uterine wall (cæsarean section).

Before entering on the indications for the operations above mentioned, the most important question for discussion, as to the treatment, is, how far, with contracted pelvis, can we count on a spontaneous labor process? This question has, since Smellie, stood in the foreground of discussion, and has only at times been somewhat obscured by the prevalence of certain operative procedures which stand in contradiction to the observation of the natural process of labor.

These were the operations in which, on the one hand, too extreme an interest for the mother was prominent (abortion) or, on the other hand, interest for the child, as in artificial premature birth and prophylactic version, operations, which, at the start, dispense with observation of the natural process of labor, and yet spontaneous birth is by far the best solution of all the many complicated questions which confront us in contracted pelvis. To be sure, it may be objected that spontaneous birth is not always possible. Unfortunately, this is true, but spontaneous birth is much more often possible than would appear from our statistics. By too hasty an interference with the natural course, however, many births are forced into a wrong track, to the great injury of the mother and the child.

As a matter of fact, at my clinic, about 80 per cent. of births with contracted pelvis take a spontaneous course, considering only births at the proper time, and there is no doubt, that if the principles which now hold good are generally and consistently carried out, the number of spontaneous births with contracted pelvis, will be further greatly increased.

Almost of itself, the question is forced on us—will not injury to the mother be brought about from this very expectant treatment, as a consequence of the long duration of labor, and the contusion of the soft parts, or to the child, in consequence of the long pressure of labor? Nothing of this kind is to be noticed. Of all possible treatments of birth, with contracted pelvis, the expectant treatment gives by far the best results for the mother, both as regards mortality, and morbidity. Parenthetically, I wish to remark that we always consider a puerperium disturbed if the temperature has risen but once to 38 degrees

Celsius. In children, also, we have the best results in spontaneous birth, as opposed to all other kinds of treatment, the cæsarean section excepted.

In regard to the results arrived at, for the mother and child, in deep forceps, version in transverse position, craniotomy with dead

THE THERAPY OF CONTRACTED PELVIS AT THE SCHAUTA CLINIC
1892 TO 1906

Number of Births—49,397

		Number	%	Mortality				Morbidity No. %	
				of mothers No. %		of children No. %			
Spontaneous births		4116	77.8	4	0.09	91	2.2	169	4.1
Indicated operations	Forceps in deep position of head.....	207	3.9	0	0	24	11.6	25	12.0
	Version from transverse position, etc...	353	6.6	2	0.5	101	28.6	32	9.0
	Extraction in pelvic presentation.....	30	0.5	1	3.3	12	40.0	2	6.6
	Craniotomy on dead child	82	1.5	5	6.0	82	100	16	19.5
	Decapitation	9	0.16	3	33.3	9	100	4	44.4
Prophylactic operations	Artificial premature birth	34	0.6	1	2.9	16	47.0	2	5.8
	Prophylactic version..	95	1.7	1	1.05	20	21.0	11	11.5
	High forceps	147	2.7	2	1.3	57	38.7	12	8.1
Surgical operations	Craniotomy on living child	76	1.4	1	1.3	76	100	6	7.8
	Caeserian section on conditional indication	116	2.1	4	3.4	2	1.7	20	17.2
	Pelvic dilatation.....	23	2.0*	0	0	1	4.3	11	47.8
Total review.....		5288	10.7	24	0.45	491	9.28	310	5.86

* Referring to the period at which hebosteotomy was first performed.

child, and decapitation with contracted pelvis, I would refer to the annexed table.

We might be inclined to accept these figures without comment since they deal only with cases which were treated according to strictly prescribed universally prevailing rules, and in which there was not

much to be changed, and yet the query will be justified, whether, in all these cases, by timely professional help, the fate of the mothers, as also of the children, might not have been shaped more favorably. In the cases of version, the question would be justified, whether by choice of another therapy more favorable to the child, the high mortality figure of the children might have been reduced. As, in one third of the cases, we had to perforate after the version, *cæsarean* section performed at the right time, or *hebotomy* might have saved these, and perhaps still other children. The same holds good of the cases of pelvic presentation. Not always in pelvic presentation have we simply to extract; in contraction of higher degree, one of the surgical operations may here be indicated, as well as also in head presentation. But many of these cases are of long ago—to-day, the objections would certainly be pertinent.

Especially of *craniotomy* on the dead child and *decapitation*, it must be said that these operations were always expressions of poor obstetrics. All these cases came to the clinic too late. Help was no longer possible for the child, and the women were, as the high mortality and a high morbidity show, often already infected before entering the hospital. An exact and up-to-date obstetric treatment from the start would have given almost all these cases another direction.

If we now turn to the important group of prophylactic operations, we have in the first place to mention the induction of artificial abortion. This operation was introduced by William Cooper in 1771, in order to evade the *cæsarean* section with absolute indication. In 1874, Cohenstein could collect only ten cases from the entire literature in which the abortion was carried out in pelves contracted to the highest degree. Owing to the safety of the *cæsarean* section, this operation has lost its justification. I have never operated from this indication.

On the other hand, the artificial induction of premature birth is held to be a fully justified operation which is everywhere practiced in contracted pelvis. It must, like the prophylactic version, be considered in the first place as a procedure undertaken in favor of the child, intended to evade *craniotomy* at the normal end.

The indication for artificial induction of premature birth is regarded as given in a conjugate of seven and a half to eight and a half centimeters in a generally contracted pelvis, and of eight to nine centimeters in a flat pelvis. As, however, we can count on sponta-

neous birth even at eight centimeters, the indication for artificial induction of premature birth is dropped (or even at seven and a half centimeters).

Nor is the artificial premature birth the small procedure which some authors would like to make it appear. If, to be sure, we had in view only in its narrowest sense the act of induction, the insertion of a bougie, the artificial rupture of the amnion, or the tamponade of the cervix, we would, of course, have to deal with a slight procedure only. But, in the further course, very serious procedures frequently come into consideration, of which it cannot always be maintained that they would have been unavoidable, if the normal end had been awaited. Not all cases of premature birth take a spontaneous course—therefore, the surgeon who counts only the cases which take a spontaneous course does not obtain a correct picture of the significance of artificial premature birth.

The high mortality of children has, with justice, given rise to the question whether we are justified in looking upon premature birth as an operation to be performed in the interest of the child.

Statistics of deliveries at term and premature of the same women, prove the value of this operation. Dohen was the first to hold this opinion. But many of the preceding deliveries belonged to a previous time, and had not taken their course under clinical direction, circumstances which may have easily brought about a change to the disadvantage of the children.

The following observation seems to me far more profitable. Let us take from the total number of our cases of contracted pelvis, those which, according to general opinion, seem particularly adapted for the induction of a premature birth, therefore multiparæ with a conjugate of seven and a half to eight and a half centimeters. If we now exclude artificial premature birth, it shows that dispensing therewith in just those cases which according to the indications hitherto set up may be regarded as predisposed thereto, the results for mother and child are not only not made worse but even slightly improved.

If, however, we do not wish at present to cancel artificial premature birth from the series of procedures with contracted pelvis, as some already do, I nevertheless believe that one should limit it to multiparæ who have previously given birth to large children with large skulls, children who are able to withstand the injurious in-

fluences of premature birth better than weakly and poorly developed children. But such children would not any longer be considered as prematurely born, judged by the degree of their development, but only by the time of their intra-uterine life. Considered from our social standpoint, it cannot be our task to bring into the world feeble and prematurely born children. We need strong and perfect individuals in the struggle for existence.

The prophylactic version must, for my clinic, be regarded as a rare operation.

If we wish to recognize the benefit which results from prophylactic version with contracted pelvis, we will, as we have done in discussing premature birth, draw into consideration, first, the total results with the prophylactic version, and then without, in the category of cases in which the greatest benefit is to be expected from the prophylactic version; these are the cases of multiparæ with a flat pelvis, and a conjugate between eight and a half and nine and a half centimeters.

From this view, the prophylactic version has made the total result worse, and that, just in the category of cases with contracted pelvis, most favorable for the prophylactic version, instead of improving as might reasonably have been expected.

Finally the comparison of spontaneous births and prophylactic version is very instructive in its results in the same categories of contracted pelvis in which the most favorable conditions for the prophylactic version; therefore, smooth pelvis in multiparæ were taken as a basis. Here also, the results deteriorate, on inclusion of the prophylactic version.

It results from these considerations that the prophylactic version has to be excluded in those cases where the probability of spontaneous birth is present.

The high forceps, considered by some authors also among prophylactic measures, does not deserve to be ranked in this group, as it is performed only on definite indication in advanced stages of labor. It is justified in cases in which, with indication of craniotomy of the living child, a last attempt for its rescue has to be made.

If we consider the cases in which, without urgent indication in the mother, the operation was performed in the interest of the child, solely, we have a fifty per cent. mortality of the children.

An operation which cannot be called an indifferent one for the mother, and in which, when undertaken exclusively in the interest of the child, only every second child can be saved, had, therefore, better be replaced by another. Only then, when the operation is in lighter degrees of contracted pelvis, between eight and a half and ten centimeters is undertaken, and in cases in which an urgent indication is present in the mother may the forceps be applied as trial, to give way immediately to craniotomy when any difficulty arises in this trial.

In addition to this operation of the prophylactic version, and the high forceps, we have now still to mention craniotomy on the living child, an operation, which in the opinion of some authors, for instance Pinard, should be completely cancelled. But for the present, we cannot yet wholly dispense with it, as an emergency operation when it is impossible to perform the competing operation. In future, its place will be taken by the *cæsarean* section, or *hebotomy*. To-day, it is still indispensable in cases which come too late under expert treatment, in advanced stages of labor, often infected, in which, therefore, a contraindication to the *cæsarean* section or to *hebotomy* is present, and in which, with high skull position, the high forceps, and in simultaneous high degree of tension, the version, are prohibited, and in which the delivery may no longer be delayed. It is, therefore, in the truest sense of the word, an emergency operation.

Craniotomy on the living child is one of the operations which in the future must be completely cancelled from the series of obstetric operations. We shall see, in the end, when, and under what circumstances, we may expect this golden age in obstetrics. For the present, this operation is to be limited as much as possible.

If, finally, we come to the discussion of the so-called surgical operations, in contracted pelvis, we have first to mention the *cæsarean* section, from absolute indication.

The mortality from morbidity in child-bed amounted to seventeen and two-tenths per cent.

These results must soon be essentially improved under the influence of more rigorous wound protection, as now customary at my clinic in laparotomies (rubber-gloves, mouth-mask, and iodine painting of the abdominal walls).

The technic of the *cæsarean* section in my clinic is the generally

practiced one, with longitudinal incision of the uterus. The transverse incision is performed in severe expansion of the lower uterine segment. For the suture of the uterine wound, silk, in two layers, is employed.

The last and most recent of obstetric operations in contracted pelvis is hebosteotomy. We prefer it to symphysiotomy. It is indicated in a pelvic contraction of seven and a half to eight and a half centimeters (*conjugata vera*) when no infection exists, and when the patient does not expressly refuse the operation.

For the performance of hebosteotomy, the same strict indications need not be demanded, as for the *cæsarean* section from relative indication. Cases which are examined previously, may, if at the time the operation is to be performed they show no signs of infection, be subjected to hebotomy, while, for the *cæsarean* section, I promise absolutely safe asepsis. I would not, however, go so far as some surgeons, who, in a manifest infection, also, see no contraindication to the performance of hebosteotomy.

We operate after the subcutaneous method of incision according to Doederlein, and complete the birth as soon as possible, since by waiting, there is danger to the child. In *primiparæ*, the operation is to be avoided on account of the danger of injuries to the soft parts.

All cases, including those of former symphysiotomies (fifty cases), took a favorable course. In forty-three and four-tenths per cent. of the cases, however, there was fever in child-bed owing to the frequent injuries to the soft genital passages. Of the children, only one died (four and three-tenths per cent. of mortality of the children).

The results of hebosteotomy, therefore, in regard to the high morbidity, are certainly in want of improvement. This improvement will ensue, especially in *primiparæ*, who wait for a spontaneous course, as has been done at my clinic only since the middle of nineteen hundred and seven, and does not yet find expression in the data at hand.

If now, in conclusion, I may be permitted to summarize the experiences, up to now, in the treatment of contracted pelvis, and to draw therefrom deductions for the future, I would express myself as follows:

The management of birth in contracted pelvis will, with expert treatment from the start, with strict asepsis, and clinical treatment, have in the future, to take the following simple course:

For cases with a conjugata of over eight centimeters, the possibility of spontaneous course of birth may be counted on, and an expectant treatment is to be pursued. For cases of under eight centimeters, the cæsarean section is to be contemplated. For the bordering cases, with a conjugata of eight and a half to seven and a half centimeters, hebosteotomy in multiparæ should be taken into consideration. This operation, therefore, would have to be regarded in the narrow breadth of eight to eight and a half centimeters as competing operation of spontaneous birth, and in the breadth of seven and a half to eight centimeters as competing operation to the cæsarean section, according, as the size and hardness of the skull, the power of labor, the abdominal pressure, and the condition of the woman in labor, indicate the one or the other management of birth. This management of labor might be regarded as the typical one of the future.

All other procedures hitherto customary in contracted pelvis are in the future to be regarded as atypical, accordingly, as procedures which are not to be regarded as lying in the nature of the interference with delivery or its obstacle given by contracted pelvis, but as accidents carried in from without which are able to influence the typical operation.

Thus, craniotomy of the dead child may be indicated in a conjugata below nine and a half centimeters. Here, the previous death of the child constitutes the atypical point. Craniotomy of the living child may become necessary in infection of the woman, or in the event of her refusal to have one of the typical operations performed. Artificial premature birth may also be performed when the pregnant woman has a disease which might make the cæsarean section or hebosteotomy at the normal end appear too dangerous, or upon decided refusal to have one of these operations performed. High forceps and prophylactic version should, if possible, be altogether struck out of the series of procedures with contracted pelvis.

High forceps might exceptionally be tried before craniotomy of the living child, and perhaps we may thus succeed in saving a child now and then.

Prophylactic version may appear justified before craniotomy of the living child, in certain degrees of simple smooth pelves, when the cæsarean section and hebosteotomy are prohibited on account of

disease of the woman in labor, and when an attempt with the forceps seems impossible on account of high and movable position.

The last two named operations, high forceps and prophylactic version, will, therefore, be indicated in such atypical cases as last attempt before craniotomy, in order to save the child's life; the high forceps in fixed, and the prophylactic version in movable position of the head.

In a conjugata under six and a half centimeters, the cæsarean section is absolutely indicated, in aseptic birth, the extraperitoneal cæsarean section with uterine suture after Latzke, with premature bladder suture, and with strong tension of the lower uterine segment, is to be taken into consideration. In uncertain asepsis, every form of extraperitoneal cæsarean section is to be *dissuaded from*, and in such cases the cæsarean section, with uterus extirpation or supravaginal amputation, is performed. The most recent study of the therapy of contracted pelvis will not change this, and therefore, I shall not enter further into this question.

In general, our efforts will be directed *to leave births with contracted pelvis to nature as long as possible*, and in case a natural completion is impossible, to perform those operations which are most certain to save mother and child. All other operations should gradually be limited more and more. But, before this "golden age" in the management of labor in contracted pelvis will come, certain conditions must first be fulfilled.

The parturient must come into the expert's hands in perfect aseptic condition, without any inadequate obstetric procedure having preceded. Outer condition must be such as to enable immediately, and strictly aseptically, any obstetric procedure. Finally, our hands must not be tied in that the parturient or those about her refuse certain operations.

All these conditions may most surely be fulfilled by demanding *hospital treatment from the beginning of labor in higher degrees of pelvic contraction, especially if the course of previous births was difficult*. Thereby, one may best observe asepsis from the very beginning, and most surely exclude all the inadequate procedures of physicians and midwives. The natural forces will be able to act with less interference than when parturient, midwives and relatives influence the physician by demanding speedy delivery. In case an

operative procedure becomes necessary, all appliances are at hand. Moreover, after long consultations, a procedure is decided upon which might be performed at home, but in which concern is principally taken of the mother.

It is undoubted that we may perform no operation whatever without the consent of the patient, be it an obstetric or a gynecological or a surgical operation. This consent is generally presupposed, and the choice of operation must be left to the judgment of the surgeon. This has long since become quite a common thing in gynecological operations. Why, then, should the parturient have the right to refuse just the operation which the surgeon, after mature consideration, considers best for mother and child? By the very request as to whether the parturient would consent to a special operation, the danger of such operation is suggested to her. Do we not perform other equally dangerous operations without special consent of the parturient? The high forceps and the prophylactic version are dangerous, too, they also may cause injuries and lacerated wounds to which any smooth aseptic incised wound is to be preferred; nevertheless, no one asks the parturient whether she consents to these operations or allows the killing of her child by craniotomy. But, as regards surgical operations in contracted pelvis, let us freely confess that we are still deep in traditions of the time when cæarean section was still a fatal operation, and that it were timely to break with these traditions.

It is true, certainly, that cæarean section and in part, hebotomy also, are still more dangerous than all other obstetric operations in contracted pelvis. If all women whose condition might demand surgical intervention awaited the onset of labor at a hospital where perfect asepsis is observed, all the incidents which now influence the cæarean section so unfavorably would be avoided. Now-a-days, women come to the hospital at all times of the day and night, often terribly neglected. In half an hour everything should be prepared for the cæarean section, the abdominal walls should be disinfected, narcosis must be given, although the patient may have taken food and drink shortly before, and the physicians, who at a late hour of the evening or night, were not prepared for a laparotomy, must quickly subject themselves to a disinfection which is often insufficient if the hands had become infected in the previous days' work. The statis-

tics of our clinic, as well as those of other clinics, show that infections and pneumonias occur under such conditions, and it is by these statistics that we judge the dangers of the cæsarean section. These statistics must be redrawn on the principles I have before pointed out; then the dangers of this operation will be reduced to a minimum, and each of us will be able, with a clear conscience, to recommend any operative procedure to the parturient. Till then, however, the danger percentage must be reduced as much as possible by a strict selection of cases.

Wholly unsuitable, and hindering all progress, seems to me, the constantly repeated indication that a physician in the country is not qualified to perform the cæsarean section, and hebosteotomy, and therefore, must continue to practice the prophylactic operations and high forceps, and that, for this very reason, the greatest stress must be attached to these operations in the obstetric schools. If the practitioner would accustom himself to recognize higher degrees of pelvic contraction in time, that is, before birth, and if he would send such cases to obstetric institutes in time, then progress might be gradually attained in the country, as well as in private practice. The physicians must use their influence on midwives, and the public, and not continue to let events take their course, and perform the old accustomed operations, most frequently, the high forceps and craniotomy, with sacrifice of the child. Numerous cases prove that a reform is possible in this direction.

It is also important, that the educational level of midwives be raised. Nothing is accomplished by *saying* that the physician is mostly called too late. We must endeavor to get this changed, and it will be changed, if but general effort be directed thereto.

CRITICAL OBSERVATIONS ON THE CURRENT PROBLEMS OF GERMAN PEDIATRICS

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It is readily apparent that German pediatrics have gained great stimulus in the last few years from experiences incident to the conditions during the war. Because of the underfeeding which involved virtually every rank of society, special attention had to be devoted to that age period during which inanition becomes ominously noticeable, namely, childhood. It became imperative on the one hand that the mothers be willing and anxious to nurse their own children, and on the other hand to assure the necessary food supply for the artificial feeding of infants and older children.

The experiences in other wars and sieges were repeated during the World War, and breast feeding increased considerably in Germany during the last years. But because of undernourishment all mothers were not able to nurse their children satisfactorily, while the low fat and albumin ration led to a decrease in the caloric value of the mother's milk, which resulted in underfeeding of a considerable number of sucklings.

At this time, reference should be made to the nursing ability of mothers in Germany. Statistics were collected under my direction in the obstetrical division of the National Institute for the Prevention of Infant and Child Mortality, where the mothers come under observation before the onset of lactation, where they are encouraged to nurse their children and are instructed in the technical details. About 85 per cent. of all mothers were able to nurse their children, the other 15 per cent. were either unable to do so or could not nurse them satisfactorily. If the mothers receive sufficient nourishment, devote their time exclusively to household duties and are not engaged in extradomiciliary labor, the same percentages will very probably apply to mothers in general. Under present conditions, however, this

ideal figure is never remotely approached and we must therefore aim to improve the methods of artificial feeding.

In speaking of an ample diet for nursing mothers, I mean that for the secretion of 800-1000 grams of breast milk, a mother should have a food allowance in excess of that normally required, the excess representing approximately 1 1-2 liters of milk. On a daily ration of 3500 gram calories a mother will easily be able to nurse a child, provided the water balance is also maintained.

In respect to artificial feeding of healthy infants we have reached the stage where the fear of overfeeding, formerly prevailing in Germany, has declined. This fear was based on the belief that many deaths in infancy were to be attributed to overfeeding, while the dominant theory regarding the manifestations of definite diatheses related these to the same cause. When I discuss the quantitative element of artificial feeding it should not be understood that I rate this principle higher than the qualitative. As a matter of fact the quantitative principle is a logical subject of discussion only when the diet contains all the requisite elements (Bausteine) for the growing organism. It is well known that the most purposeful method of estimating the dietary requirements is by calories, and from 100 to 120 calories per kilogram of body weight are considered necessary in artificial feeding as well as breast feeding. This is a precise statement of the principle of minimum nutrition which we have followed until now. Latterly the subject of concentrated feedings has been discussed to an extraordinary degree in Germany. As the calculations will disclose, two things are meant by the term: A diet containing the same absolute quantity of food substances with a relatively smaller proportion of water, and a diet which provides a greater quantity of food units. It is claimed for this concentrated diet that it gives greater assurance of normal growth and prevents the development of a series of disorders, *e.g.*, rickets and certain manifestations of the exudative diathesis.

Heretofore I have not seen any outstanding benefit of water-poor feeding under physiologic and pathologic conditions, and I see no advantage in rigidly restricting the water intake of the infant, whose body contains a greater percentage of water than any other organism. On the other hand, I need only refer to the ever increasing experiences which demonstrate that thirst and water deprivation are especially

deleterious to the child and are responsible for a number of pathologic phenomena. Thus there is lately an increasing tendency to attribute the transitory fever of the newly born to thirst, a belief which I myself maintain on the basis of experimental studies, while many authors ascribe a not inordinate rôle to thirst in the intoxication complex. On the other hand I will admit that a number of infants are not injuriously affected by restriction of water intake, such as is the rule in concentrated feedings, and that, in fact, they may even show excellent development. With the aid of concentrated feedings it is possible to avoid the danger of hunger without the danger of approaching exsiccation, especially in children who do not take liquids readily or who have little or no appetite. I recognize a marked advantage of concentrated feeding in this fact only, but to my mind it is the only advantage. As I remarked in the introductory sentences, in the German literature concentrated feeding does not necessarily mean a low water intake but it can readily be determined that occasionally it signifies overfeeding and fattening (Mast), to which excellent results are attributed contrary to the prevailing opinion of former times. Without reference to the possible harmlessness or even benefit of fattening of infants in certain indications, especially under pathologic conditions, I do not recommend it for the healthy infant but I would counsel instead that the principle of minimal feeding be followed and that a diet of approximately 120 calories per kilogram of body weight be maintained. There was no marked increase in infant mortality or nutritional disorders during the war, when the means of artificial feeding were extraordinarily limited and rationing had to be enforced for infants also (it was impossible to enrich the infant diet with fat, the milk had low fat content, and the prevalent shortage did not permit more than 30 grams of sugar and 20 grams of flour for each child). Apparently the principle of minimal feeding was extraordinarily beneficial and the group experiment which the conditions of war imposed should be our guide in times of peace and should deter us from giving approval to fattening or overfeeding of infants.

From the fact that a considerable number of infants thrive on rich feeding it is evident that in a large percentage of cases the powers of assimilation vary within wide limits and that the dangers of artificial feeding are not of real moment to all infants. On this point, too,

experiments with concentrated feeding have yielded some very interesting disclosures, namely, that carbohydrates as well as fats are tolerated in high concentration. The studies of Pirquet and his pupils on carbohydrates have taught us to prescribe mixtures with high percentage of carbohydrates (up to 17 per cent.) especially for the newly born. We became cognizant of this fact in relation to fats through the practice of thickening skimmed milk with a butter and flour mixture, the so-called butter-flour feedings (*Buttermehlnahrung*), which proved effective in a large number of cases.

Naturally there can be no discussion regarding the applicability of these various food mixtures—some enriched with carbohydrates, others with fats—as so-called standard artificial foods to be indiscriminately prescribed for all infants. There is no artificial food which is suitable for all infants like breast milk is, and to none can we attribute those qualities which differentiate natural feeding from all other methods: The avoidance of nutritional disorders, severe forms of the English disease, spasmophilia, rickets, and the increase of immunity. It can simply be stated that good results may be expected of a large series of variously constituted artificial mixtures and that the correlation between the food elements and mineral salts is of a decisive importance in determining the suitability of any food, a fact to which I originally referred twelve years ago.¹

This correlation depends on the relation of the carbohydrates to the fats, on the relation of albumin to salts, etc., and different infants certainly require different correlations as most beneficial to their individual needs. With a given quantity of fat and albumin a very definite quantity of carbohydrates is necessary, and for a considerable number of children this will be much greater than that found in the ordinary mixtures, while other children require less carbohydrate food. It cannot be maintained off-hand that albumins represent the constipating principle and carbohydrates the diarrheal factor of the diet, for this applies only for certain correlations of the food elements and for certain definite children.

If in a given food the requirements of albumin, fat and carbohydrate (the latter varying within wide limits) are satisfied, the most varied correlations can evidently render excellent service. As has

¹ Festschrift bei der Eröffnung des Kaiserin Auguste Victoria Hauses.

been stated before, it is necessary that every food element (including the mineral salts) be present in the minimum quantity required for normal development. This was apparently not always the case in the old popular formula of artificial feeding—the one-third mixture—and for that reason the artificial feedings are now started on half milk, a rule that is quite general throughout Germany. As a result we no longer meet with cases of underfeeding and inanition, which occupy as prominent a position to-day in the evaluation of nutritional disorders as overfeeding did formerly. The addition of carbohydrates is on a more liberal scale, never less than 5-6 per cent., the ratio of farinaceous material to sugar being approximately 1:4. Pirquet and his coworkers have taught us that much larger quantities of carbohydrates are tolerated or perhaps even required by many children. In this connection let us be reminded that a mixture of whole milk with 17 per cent. of sugar figures among the food mixtures recommended by him.

The carbohydrate requirement of infants evidently varies to an extraordinary degree, and every mixture, especially as related to the percentage of fat, needs a different proportion of carbohydrates. By no means, however, should this principle be pushed to exaggeration, for an excess of carbohydrates may lead to habitual vomiting, of which fact I became convinced in personal tests of Pirquet's system.

The albumin of cow's milk is of far greater importance than was formerly assumed. Without albumin there can be no growth of the infant, in fact the organism will be seriously impaired. Besides there are certain definite relations between albumin and the bacterial flora of the intestines. Without further evidence we can place albumin among the complimentary food stuffs, in the absence of which the child will not maintain satisfactory growth.

Provided that in a given artificial mixture there has been taken into account the infant's needs of the elements necessary for maintenance and growth, the quantitative principle assumes the same overwhelming importance as in the dietetic management of adult persons. In Germany, as a rule, the quantitative estimations are still made on the basis of calories. We must abandon the belief that in artificial nutrition an energy quotient of 100 calories per kilogram of body weight suffices for every individual child and applies to every food mixture. For the variation is just as extreme as in the case of adult

persons, depending on the character of the metabolic processes and the temperament of the child, while some food mixtures will have a greater nutritive effect than others. It should probably be stated then that the energy quotient in artificial nutrition may run as high as 125-130 calories, although some infants thrive on 100 calories or less. In spite of these variations a certain norm can be established for the practical guidance of the physician. In Austria, especially in Vienna, under the influence of Pirquet, the caloric system has been abandoned and in its place the "nem," representing 1 gram of mother's milk, has been established as the unit for the estimation of the nutritive requirements of infants. On this basis Pirquet has built up a detailed, carefully planned system of feeding, which was applied on a large scale during the war in the rationing of the undernourished children and in the application of which the present generation of physicians and nurses is being educated. The system in all its phases has been rejected in Germany, perhaps not with full justice, for many excellent and important ideas are buried in the anthropometric studies which were conducted in combination with the new system. On the other hand, it cannot be denied that the same estimations can be made by calories and that the new system merely introduces a novel mode of expression, though facilitating, it is true, an accurate concept of quantitative feeding among great masses of people, for which Pirquet deserves special credit.

If we consider the newer views concerning nutritional disorders, it is apparent that these are all still in a state of flux, although, after all, the clinical point of view is in the foreground and the bacteriologic era is fast displacing that in which attention was riveted on the food substances. To-day the pathogenesis of acute digestive disorders is attributed to the colonization and proliferation of bacteria within the small intestine under certain pathologic conditions and under the influence of impaired resistance, whether produced by a dietetic error, infection, carelessness in nursing, overheating or exposure to cold. From this moment there are signs of a disposition to dyspepsia, such as abnormal changes in the chyme, inflammatory irritation of the intestinal mucous membrane, absorption of abnormal products of digestion or bacterial products into the blood, producing the clinical picture of dyspepsia and intoxication. There are many theories regarding the peculiar reaction of the infant

organism with the toxic symptom complex, and while various authors have different conceptions about its principle phases, discorded consciousness is always in the foreground. It is possible that the acidotic constitution—so-called by Ylppö—gives rise to this peculiar reactivity. It remains undecided what influence is exerted by the exsiccation of the organism, by the absorption of bacteria, endotoxins, or the higher-split products of proteins in the blood.

The theories regarding the pathogenesis of chronic digestive disorders are not so clear cut. In my opinion, however, a definite advance was marked by the fact that Finkelstein has abandoned the expression of "disturbed balance," and has substituted the clinical phrase of "dystrophy," corresponding to my designation of "hypotrophy." This term will probably give way to "decomposition," as physicians have a fixed idea regarding the clinical picture of these disorders. It should be added here that questions of nomenclature and theoretic speculations are partly responsible for the fact that many practitioners are not versed in the diagnosis and treatment of chronic digestive disorders, or fail to apply the advanced knowledge gained by practical experience. To me it seems specially important to the interests of practical infant welfare that this chapter be divested of superfluous theory so that the subject may be made as simple as possible, and for this reason I offered certain suggestions and presented a scheme in a previous article (1). In the first place it is very necessary that the medical practitioner differentiate between mild and severe disorders, so that he may determine when it is safe to use artificial mixtures and when it is imperative to substitute the cardinal health food, breast milk. Despite our uncertainties regarding the pathogenesis of chronic digestive disorders, we have undoubtedly made extraordinary progress in the treatment of these diseases. First of all, we now appreciate the importance of inanition in these cases, and therefore avoid unnecessary fasting. As soon as possible the disturbed organism must be supplied with all requisite food elements (Bausteine) in sufficient quantity and in a mixture that will be readily assimilable, permitting normal cell growth, an essential basis for normal water fixation. Naturally, breast milk best fulfills these requirements, for although success with a given artificial mixture may have blinded certain authors, the idea is gaining ground daily that breast milk is not only the best food to maintain the health of an

infant but also to restore it in cases of disordered nutrition. However, we must remember that in certain severe conditions of inanition, especially in various forms of atrophy, breast milk must be supplemented by an artificial mixture rich in salts and albumin, avoiding the development of a predisposition to dyspepsia. Either buttermilk or milk and egg albumin, with a definite amount of carbohydrates (the dextrin-maltose mixture, 3-5 per cent., being perhaps the best) may be prescribed. In general, I am opposed to fixed dietetic prescriptions in the sense that the practitioner need only know how to employ ordinary trade preparations. He should be able to improvise mixtures without reference to the pharmacist, and this should be based on a definite knowledge of what he seeks to attain. He should be able to proceed from the known fact that a mixture high in carbohydrates but low in proteins readily induces diarrhoea, but that more carbohydrate is tolerated in a vehicle enriched by albumin.²

The dangers of inanition are especially serious in the dysenteric disorders of children which have lately increased considerably instead of the ordinary summer diarrhoea. A number of children with this affection succumbed to the gruel mixtures continued over weeks at a time, and not to the dysentery toxin or the bacterial invasion of the large intestine. They might have been saved if after thorough evacuation and irrigation of the intestines a more nutritious diet had been ordered. In these cases, the general condition, especially the nervous symptoms, and not the stool findings, should decide the dietetic regimen and it is absolutely wrong to continue gruel feedings so long as the stools show traces of blood. The mortality rate of typhoid fever has declined since we allow a more nutritious diet, and the same holds true in dysenteries, and a knowledge of this fact by the majority of physicians will assure the recovery of many children who would otherwise succumb to the effects of protracted feedings with gruel mixtures.

With this I will drop the discussions of the nutrition and disorders of infants and will consider three diseases which have again been forced into the foreground of interest by the war and incident conditions, namely, rickets, syphilis and tuberculosis.

We have latterly become acquainted with the severest forms of

² LANGSTEIN: *Deutsche med. Wchnschr.*, 1921, xxiii, 10. LANGSTEIN-GÜPPELT: *Ueber Prophylaxe und Therapie der Kinderkrankheiten.*

the English disease. The cause of this lay in the food blockade, for it is common knowledge that infants and small children react rapidly to deficient or unsuitable feeding by pathologic bone development, though adult man is not immune to the same reaction. We have seen cases of the fragility of the bones the like of which had never come under our observation before, though this fact has by no means led the profession in Germany to accept the point of view that rickets is simply an avitaminosis. Even though we were not in a position during the war to study the vitamin question with the intensity with which this was done in America and England, there are at our disposal a number of valuable and critical works that permit us to state that the conception of rickets as an avitaminosis has not solved the problem but has given it a one-sidedness which may prove detrimental to future investigations. The youthful bone reacts to the most variegated unfavorable influence, and invariably through changes of the osteoid tissue. This reaction is not limited to a certain dietetic regimen, but we must also consider the hygiene and care of the child.

The treatment of rickets has been the subject of many experiments and a distinct advance has been made by the fact that artificial mountain sunlight can affect a cure of the severest forms of osseous rickets in a relatively short time. This domain of artificial heliotherapy is uncontested. So far as the question of diet is concerned, we still maintain the position that a moderate amount of milk with carbohydrates, vegetables and fruit constitutes the most serviceable regimen. Perhaps the presence of accessory food factors in this mixed diet plays a definite rôle in the cure of rickets. It seems to me to be premature to exploit commercially the therapeutic value of these accessory food substances in rickets and Barlow's disease (to mention only the principal diseases), and to put vitamin preparations on the market, as has been done in Germany and perhaps even on a larger scale in other countries.

Congenital syphilis has also increased during the last few years. The methods of therapy have been greatly improved inasmuch as we no longer consider any treatment sufficient unless from four to six courses have been given during the first two years of life. The combined mercury-salvarsan treatment is in greatest favor and on all sides emphasis is placed on the necessity for constant serologic control of all cases during treatment. A sufficient number of years has

not elapsed to determine definitely what percentage of cases of congenital syphilis is permanently cured and what method is best. The fact, however, that a large number of patients, even with visceral syphilis, after thorough treatment have been free from symptoms of the disease for almost a decade and persistently show a negative Wassermann reaction justifies the optimistic view that this scourge will eventually be conquered when the public has been properly educated.

Finally, in regard to tuberculosis, it must be admitted that this disease has increased to an alarming extent. Heretofore, as a rule, children of the proletariat were not infected until about the fourteenth year of life, to-day the majority of young children harbor the tubercle bacillus, at least in the lymphatic glands. The importance of this infection is therefore much greater for our children and the chances of a victorious outcome in the fight against glandular tuberculosis are decreased.

The disease has increased in frequency not only among the children of the proletariat, the same holds true of the middle classes and the well-to-do. This is disclosed by the results of biologic tests, among which the intracutaneous reaction alone is acknowledged as trustworthy in Germany. Because of the many sources of error the röntgenogram must be studied with great care.

The early diagnosis of tuberculosis is of primary importance in children. A nutritious diet with complete satisfaction of the requirements of albumin and fat and avoidance of carbohydrate fattening is absolutely necessary, along with rest, fresh air and sunlight. We have learned lately that heliotherapy can be successfully applied even in the lowlands, on house-tops and in the open field, and that it is by no means necessary to send a child to the mountains in order to cure tuberculosis of the bronchial glands.

Divergent views are held in regard to the efficacy of tuberculin. I have no doubt that investigation will gradually reveal what beneficial factors are inherent in tuberculin therapy and will determine the proper indications for the selection of this preparation. The hopes placed in Friedmann's remedy have not been fulfilled, but there is no ground for the pessimistic view that a specific for the disease will never be discovered.

Industrial Medicine

INDUSTRIAL SURGICAL CLINICS

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WORKMAN'S BACKACHE

A WORKMAN with a lame or painful back is in a serious position. He knows he has a pain in his back that prevents him from working, but often he has little else to prove this than his own statement. He is depressed by the fear of a permanent disability and this depression grows during the slow progress of his condition and his enforced idleness. He is often told by the doctor and by his employer that he is faking and this adds to his general fear that he will not be able to work and that he will not be able to collect the compensation that is justly due him. Consequently, most of our back cases come to examination with the idea that they must exaggerate their symptoms to get any recognition from the surgeon, feeling that if they acknowledge to their employer that they can do even light work, they will be denied compensation.

Many physicians, especially in the smaller places, with lack of proper diagnostic equipment, diagnose cases of lame back, unless there are marked and visible lesions, as malingerers. Many of these cases spend their own money for various cures and often get relief from someone who gives them massage or manipulations, and this only adds to the surgeon's belief that the man is faking, while in reality he had some injury, such as sacro-iliac subluxation, and an osteopath has

performed the proper manipulation and given him relief. The general subject of our diagnosis and treatment of back injuries in workmen has, in our opinion, thrown more discredit on the general medical profession than anything we see in this Clinic.

Lame backs have a bad reputation with insurance companies and employers. This, the medical profession should realize, is their fault, for the insurance companies and employers accept the word of their attending physician that the man with the lame back is faking. Consequently, lame back cases form a large percentage of the compensation cases heard before industrial boards at this time.

The members of the compensation boards have seen many back cases diagnosed as malingerers by the sworn testimony of the company doctors. The same cases have been examined by their medical examiner who usually has availed himself of hospitalization and a large diagnostic equipment, and has often found in the so-called malingerers, extremely serious back injuries or some disease that may or may not be due to an injury, but should have been explained to the man and treated long before. Therefore, the members of the Compensation Board view the medical testimony in back cases with great suspicion and commonly believe that if a man who is earning twenty-five to one hundred dollars a week is willing to stay off of that work for a year or more at twelve dollars a week that he has an injury, especially if he introduces some medical testimony that he has some injury. Back cases are oftentimes awarded total, permanent disabilities when they are only partial disabilities, and would have been doing some work if the case had been handled promptly and properly. The causes of back pain are many and the diagnosis is often impossible without prolonged study.

The authors have found that there are malingerers claiming back injuries that do not exist and faking inability to work after a real injury, when they are able to work; but we have also found that in men coming before the industrial boards for examination, the malingerers of back injuries are less than in other classes of cases.

In our Clinic the most mistakes in diagnosis that we have seen come from dim and blurred X-rays of the spine. In view of the above conditions we believe it would be valuable to include in this, the last issue of the Clinics, our methods of diagnosing back pain. Without

proper diagnosis, the treatment is empirical. Often to make a diagnosis of back pain we need all the modern laboratory equipment and the most skillful X-ray technic and interpretation.

PRACTICAL MECHANICS OF THE SPINE

We believe that before an intelligent physical examination of the spine can be made there must be a thorough understanding of the practical anatomy and mechanics of the spinal column.

Curves.—In the quadruped days the spine was supported at both ends; there was no cervical or lumbar curve. When the quadruped assumed the attitude of the biped, in order to maintain the upright position and eliminate excess strain on certain groups of muscles so that the weight would be distributed equally on each side of a perpendicular line which can be called the weight line, establishing easy balance, it was necessary to develop cervical and lumbar curves. In the young infant these two curves are not present. In the new-born infant the head is dropped forward, the spine has one long, backward curve, due to muscular weakness. In the child, the cervical curve appears first as muscular power is developed, to enable the infant to raise and control the head. The lumbar curve does not appear until the child stands, and the erector spinæ muscles hold the body upright against the resistance of the iliopsoas muscles and pelvic and hip ligaments.

The weight line of the spinal column would be indicated by a line dropped perpendicularly from the odontoid process of the axis with the individual in erect posture. The normal curves of the spine are four. The cervical is concave backward and begins at the first cervical and ends at the second dorsal. The dorsal curve is convex backward, beginning at the third dorsal and ending at the tenth dorsal vertebra. The lumbar curve is concave backward, beginning at the eleventh dorsal and ending at the lumbo-sacral articulation. These three curves are the effects of balancing the body in the upright posture, and are partly produced and kept up by the intervertebral disks. The fourth curve is the pelvic or the sacro-coccygeal which is a fixed curve.

These curves are altered by disease and much more frequently by occupation or postural defects.

Movements of the Spine.—The movements of the spine are flexion and extension, *i.e.*, movements of the body forward and backward; lateral motion, *i.e.*, movement of the body to the side; and rotation, *i.e.*,

twisting the body. Flexion and extension are greatest in the cervical and lumbar regions. Motion is very limited in the thoracic region, as it forms part of a rigid chest wall, the spinous processes overlap and the intervertebral disks are thin. Lateral motion is greatest in the neck, as will be understood if we consider that the articular processes face up and down more than in any other direction.

Muscles.—The flexors of the spine are the muscles situated in the front of the vertebræ, and it is not essential that we should know the names of the various groups. The flexors of the spine are practically never strained by injury. These muscles are brought into play if we have a patient lying flat on his back on a table and direct him to rise from this position to the sitting one. In this motion the pelvis is first fixed by the action of the thigh muscles. The head is lifted from the ground by the action of the muscles in front of the neck. The dorsal region is then raised by the anterior abdominal muscles. At this time the normal lumbar curve has disappeared; the rectus and other abdominal muscles continue to act, assisted by the flexors of the spine. The psoas and iliacus assist in this flexion and tilt the pelvis around the axis of the hip joints until the sitting position is reached. The further flexion is now restricted by the ham-string muscles, which, as the knees are extended, are fully stretched. As the knees are bent further forward flexion can apparently be obtained, but this is only apparent as the movement is most entirely obtained by rotation of the pelvis around the hip joints, as the ham-string muscles are now relaxed.

The extensors of the spine are situated on either side of the spinal column. The largest and most important of these muscles is the sacrospinalis, often called the erector spinæ. This muscle is the most frequently strained back muscle, as it does most of the work and heavy lifting. It is readily palpated, as it is just to the outside of the spinous processes just under the skin and posterior layer of the lumbar fascia. In the attachment of the muscles of the back, as in muscles elsewhere, there are two forms of attachments. First, the tendons of origin may be inserted into the muscle fibres gradually, or second, these fibrous tissue bands may be inserted abruptly. Where the junction of fibrous tissue of origin into the muscular tissue is gradual, the means of junction is strengthened. Where there is no gradual imbedding of fibrous tissue with muscle, there is a weak spot, as muscle tissue possesses more elasticity than fibrous tissue. One of these points is where

the extensor muscle is attached to the transverse band extending outward on the transverse processes of the lumbar vertebræ and there is likely to be a strain at this point. The sacro-spinalis muscle runs between two layers of dense fascia, the fascia lumbo-dorsalis, which forms a sort of tunnel or tendon sheath for it.

Lumbar Region.—When the individual is in an upright position, the junction of the fifth lumbar with the sacrum is at an angle of forty-five degrees, with a downward and forward thrust at every step. In consequence of the lumbar curve there is also a distinct change in the position of the sacrum as regarding the ilia; that is, when we are sitting, the sacrum is undoubtedly the keystone of an arch, the wide part being at the top and the narrow part toward the coccyx at the bottom, but when the patient resumes the upright position the sacrum is tilted forward and downward. Now, instead of the top being above, the top is in front; the front is below; and the back is above. The back of the sacrum is narrower than the front, and consequently instead of the keystone of an arch set between the ilia, there is an inverted keystone. Now, if the lumbo-sacro-iliac ligaments are severed, the sacrum would drop forward and downward until it reached a point of rest on the pelvis, because there is no bony support except for a few irregularities on the articular surfaces of the sacrum with the ilium to support this joint between the ilia. We have then, to depend entirely on the ligaments which hold together the ilium, the sacrum and the fifth lumbar, for our upright standing position. The sacrum and fifth lumbar form a fulcrum at which all the weight from the upper part of the body is transmitted to the lower part of the body. This weight might be carried normally without trouble inasmuch as these ligaments are amply able to hold the weight put upon them when everything is normal. We know, however, that the lumbo-sacro-iliac region varies greatly from studies of X-ray findings. Dr. Archer O'Reilly, of St. Louis, has made numerous dissections of this region, preceded by X-rays, and while his investigations are not yet complete, according to his own statement, there has been proven a wide range of variation in the normal of this region.

Roughly speaking, the cervical articular facets are set almost in the horizontal plane, becoming a little more perpendicular as the base of the cervical region is reached. The dorsal roughly set in a perfectly lateral plane, that is, at right angles to the anterior posterior

plane. The lumbar ordinarily set at an angle of 45 degrees to the anterior posterior plane.

This angle in some individuals is almost 90 degrees, and in others much less than 45 degrees, the articular facets being set almost in an anterior posterior plane. When this occurs, a downward and forward thrust of the weight above falls entirely on the ligamentous support along the vertebral column. Normally the lower articular facet of the vertebra above lays behind the upper articular facet of the next lower vertebra; the lower facet facing inward and the upper facet facing outward. This has a marked effect in preventing forward slipping of one vertebra upon the other, locking them in place.

If, however, these facets lay in an anterior posterior plane, it can be readily seen that there would be nothing except the ligaments joining the two vertebræ to prevent the forward slipping of one vertebra upon another; and in the lower lumbar region especially the junction of the fifth lumbar with the sacrum, it is not an uncommon thing to actually find the fifth lumbar slipped forward on the sacrum, the finding being made by a lateral X-ray. If all these facts are taken into consideration and it is then shown that the pelvis, in a standing, forward bending position is fixed in a definite position by the ham strings which are attached to the lower posterior part of the pelvis, namely the tuber ischii, with the body leaning forward, that we have here a lever of the first class with the spine above the lumbo-sacral region as one end of the lever; the ilia and the lower extremities as the other end of the lever, with a fulcrum at the junction of the fifth lumbar with the sacrum and the sacrum with the ilia. Granting that all these structures are normal, there is sufficient factor of safety in the structure of the ligaments and bones to withstand any ordinary amount of strain. We, however, are not dealing always with the normal. We have here a group of bones and joints and ligaments which are subject to all the ills to which other bones, joints and ligaments in the body are susceptible, namely, overstrain, toxemias, poor intestinal or renal elimination, overexposure, infections, overwork or fatigue; and, therefore, we cannot assume what we ordinarily have been assuming—that it is impossible to seriously injure these structures mechanically. We know that joint pains in all parts of the body are frequent as a result of toxemias and infections and overstrains. Ligaments are poorly supplied with blood because they are connective tissue structures and

need comparatively little blood supply. Normally they are elastic to a considerable degree and we know that when they are overstrained they give sharp and severe pain. We know that they lose their elasticity when inflamed. We also know that in these structures infections and toxæmias, especially chemical deposits, occur with a greater degree of frequency than in the tissues which are better supplied with an active circulation. Therefore, we can assume in this great collection of ligaments in the lower spine, subjected as they are to great strain, that we may have severe pain as the result of a considerable number of causes. It has been definitely proved by obstetricians that in the physiological condition of pregnancy, the pelvic inlet and outlet can be increased or decreased by hyperflexion or hyperextension of the thighs. If the sacro-iliac ligaments under these conditions can relax sufficiently to allow movement enough to actually increase or decrease the pelvic inlet or outlet, then, certainly under other conditions this same relaxation can exist and allow abnormal motion. Assuming, then, that the body is bent forward and either by a lift or a sudden strain of any kind, causing sudden weight to be exerted on the shoulders, there is a tremendous strain thrown on the support around the fulcrum; and if the ligaments and muscles are not strong enough to retain the bones in their normal positions, something must give way and undoubtedly does give way much more frequently than we have previously thought.

There has been much dispute because the X-ray does not show actual dislocation. If it is remembered that X-rays are only shadows and that they have no third dimension, and although a stereo is supposed to be a third dimension, it is an extremely poor third dimension, because there are so many variations in the angle at which the X-ray may be taken and the normal varies so greatly that even with the expert a slight displacement might be overlooked, and with the average, certainly is overlooked in a large majority of cases. Attention is also called to the fact that these displacements are probably not gross enough to be detected without very critical study of an X-ray, if then. It is probable that these are rotations on an axis, the axis being a line drawn transversely from side to side through the middle of the sacrum in the case of the sacro-iliac, that the upper part of the sacrum moves slightly forward and the lower part moves slightly backward, thereby putting an acute strain on what is probably, at the time the subluxation

occurs, an already overstrained or somewhat abnormal ligament. This acute strain brings on sharp pain and the pain persists until one of two things happens; either the strain is removed by a replacement of the bones in their normal position, or the ligaments become adjusted to their new position and gradually the traumatic inflammation, which is set up by the overstretching, subsides.

VARIATIONS IN THE FIFTH LUMBAR VERTEBRA

It is extremely difficult to say what is absolutely normal in the fifth lumbar vertebra. There undoubtedly exists, however, some variation which causes or subjects the patient to greater liability of lower back pain. The position of the articular facets has already been spoken of.

There are several types of fifth lumbar vertebra.

First, one which is of goodly size and sets atop the sacrum in a proud position, with the transverse processes free, with one to one and one-half centimeters between their tips and the crests of the ilia at the nearest point.

Second, there is another type, lying low and flat between the ilia with the lateral processes projecting and overlapping the shadow of the ilia. This approaches the so-called sacralized fifth lumbar.

Another type is the actually sacralized fifth lumbar vertebra in which the fifth lumbar vertebra forms an integral part of the sacrum, lying between the ilia and being joined to the sacrum by a bony bridge; and in some cases actually joined to the ilia.

Still another variation occurs in the half-sacralized fifth lumbar, in which one side of the fifth lumbar seems to be joined to the sacrum and the other half free as in the normal. In many cases, there will be found on the side joined to the sacrum a not only long, but very broad fifth lumbar lateral process in the form of a bat wing. These are found in individuals who have no back pain as well as individuals who have back pain, as is the case in all these variations. (See Fig. 1.)

In any of these there may be what may be compared to a cervical rib in the upper spine; that is, a very long transverse process on one or both sides. In a number of cases which have come to the authors' attention there is an actual joint formed between the transverse process of the fifth and the inner surface of the ilium on one or both sides, with actual joint formation and ligamentous support. It was believed formerly that these long processes in themselves gave pain. This,

FIG. 1.



X-Ray by Dr. H. E. Potter. Long lateral process on fifth lumbar vertebra with sacralized fifth and attachment of fourth to fifth lateral.

FIG. 2.



Cervical ribs discovered by X-ray after persistent cervical pain following slight trauma.
(X-ray by Dr. H. E. Potter.)

FIG. 3.



Congenital anomaly of the first dorsal vertebra consisting in the existence both on the right and left sides of an articulating rudiment which projects beyond the end of the transverse process. This rudiment which is not more than three-eighths of an inch long appears to interfere somewhat with the articulation of the tubercle of the rib to the transverse process.
(X-ray by Dr. H. E. Potter.)

however, is not the authors' opinion, since it has been seen in a number of patients who had no pain which could be attributed to this condition; and one case which came to operation in which the processes appeared in the X-ray to be the same on both sides, but on one side only was there pain which could be attributed to the long process. This case came to operation and it was found that on the side the patient had pain, the symptoms of which will be taken up in the question of differential diagnosis, that there was a pinching of the fifth lumbar nerve, which was caught between the lateral fifth lumbar process and the sacrum in spite of the fact that normally the nerve comes down in front of the lateral processes.

Upper Lumbar.—At the junction of the dorsal with the lumbar region, we have mechanically, a hinge between a comparatively fixed and a freely movable part of the spine, there being practically no flexion in the dorsal spine. With a sudden force which drives the body into flexion, the weight of the blow will be practically entirely received at the dorso-lumbar junction. If this force is sufficient to cause an injury of greater severity than a sprain in this region, there is apt to be a fracture or a fracture dislocation in this region and the reasons for the frequency of this fracture dislocation are well summarized in Piersol's "Practical Anatomy," as follows:

The latter accident is usually caused by extreme flexion of the spine, and of the three points mentioned it is most often found in the segment including the lower two thoracic and the upper one or two lumbar vertebræ. This is due to the fact that (1) this segment has to bear almost as much weight as the lumbar spine, and yet its vertebræ are smaller and weaker. (2) The transverse processes are short, while the longer ones below, together with the crest of the ilium and the ribs above, give a powerful leverage to the muscles that move the region in question. (3) It is the region at which the most concave part of the thoracico-lumbar curve is found, making the "hollow of the back" and corresponding to the "waist" where the circumference of the trunk is smallest. (4) Its nearness to the middle of the column enables greater length of leverage to be brought to bear against it than against any other part. (5) The different segments of the spine above it are comparatively fixed (Humphry). These anatomical facts account for the frequency and severity of the injury known as "fracture dislocation" in this region as a result of extreme flexion.

HISTORY

One of the most important aids in the diagnosis of back pain is a detailed history, and we are giving here in detail our outline for taking this history, as we believe that every point mentioned in this history has had at some time or in some case a bearing on back pain.

Chief Complaint.—Under this it is wise to go to great pains to record as minutely as possible, in the patient's words, the history of the back pain; and it should be noted whether the pain is constant, dull; severe and constant; throbbing or burning. It should be noted whether the pain is referred or whether it is localized at all times, and what conditions, such as weather, influence this pain.

Present Illness.

- (a) Manner of onset of the pain or a detailed record of the accident. In this description the man should be asked to place himself in the exact attitude he was in at the time of the accident; the date and exact hour of the injury should be noted; what the patient was doing at the time of the start of the pain. If the pain was due to violence from without, the exact nature and direction of the force should be ascertained; whether the violence was a sharp blow or a continued force and the direction of this force.
- (b) Possible cause, as related by the patient.
- (c) Subsequent history in order.
- (d) Present symptoms—character of; frequency; occurrence.
- (e) Previous treatment; idiosyncrasy for drugs and food.
- (f) Symptoms in other organs and systems—
 - Cardio-vascular
 - Respiratory
 - Gastro-intestinal
 - Genito-urinary
 - Nervous

Past Medical History.

- (a) Birth—character of.
- (b) Infancy—nutrition; feeding.

(c) Diseases of early childhood—

Measles	Chickenpox
Scarlet Fever	Mumps
Diphtheria	Smallpox
Whooping Cough	

(d) Subsequent diseases—

Typhoid	Tonsilitis
Chorea	Rheumatism (character of)
Pneumonia	Colds (character of)
Pleurisy	Coughs (character of)
Convulsions—unconscious attacks.	

(e) Subsequent injury.

(f) Operation.

(g) Venereal disease.

(h) Menses and obstetrical and gynecological history.

Social History.

(a) Nationality

(b) Occupation—type of; outdoors.

(c) Personal hygiene: Housing conditions, air, ventilation, drainage.

(d) Diet—amount, regularity.

(e) Personal habits: Exercise, bath, tobacco, coffee, alcohol.

Family History.

(a) Father, Mother, Sisters, Brothers.

(b) Grandparents

(c) Husband, Wife, Children.

(d) Family Diseases, i. e.,

Renal, Cardio-vascular, T.B.

Malignancy—Gout

Rheumatism—Convulsions

Mental—Lues

The following physical examination is quoted in detail, as we believe that by following a detailed form of physical examination, nothing will be missed, and one point missed in this physical examination may be the point that is the controlling factor in the case of the back pain. We believe that it is important to emphasize the fact that the patient should be examined stripped, as in no other way can anything like a correct examination be made of cases of back pain. One

of the important things to notice is the action of the man when he is undressing himself, as a man undressing himself will perform, if he is following the ordinary custom, all the spinal motions, and malingerers very often forget this if they believe they are not being watched while undressing.

Physical Examination

1. General—

- (a) Blood-pressure, pulse, temperature
- (b) Dress and behavior
- (c) Height and weight
- (d) General build, i. e., adipose, muscular, bony
- (e) Cachexia—Wasting, i.e., T.B., diarrhœa, malignancy
- (f) Posture and movements
- (g) Station and gait
- (h) Pallor—cyanosis—jaundice
- (i) Œdema—face, extremities
- (j) Glandular enlargement
- (k) Eruption and rashes

2. Special—

- (a) Head and face: Size, shape, expression, tumor, swellings, color, movements of muscles, hair, color scalp.
- (b) Ears: Waxy, discharge, deaf
- (c) Eyes: Ptoxis; paralysis of muscles
Exophthalmos—sclera—cornea
Pupils—reaction to light and to accommodation
- (d) Nose: Septum—breathing—adenoids
- (e) Mouth: 1 Lips, color, herpes
2 Buccal mucous membranes—color, eruption
3 Teeth—dental, pyorrhœa
4 Tongue—tremor, deviation, coated
5 Pharynx—tonsils, palate
6 Larynx—voice, speech
- (f) Neck: General contour, shape, musculature
Pulsations, gland enlargements
Tracheal tug, thyroid
- (g) Chest:
 - 1. General: (a) Symmetry; standing, sitting.
 - (b) Shape, phthisis, barrel, rachitic.

- (c) Expansion.
- (d) Tumors.
- (e) Veins.
- 2. Special: (a) Lungs—
 1. Inspection, retraction, expansion, atrophy, flattening.
 2. Palpation, dropping shoulder, rigidity of muscle, muscle expansion, tactile fremitus.
 3. Percussion—Resonance, definite border, apex retraction.
 4. Auscultation—
 - (a) Breathing, type of.
 - (b) Voice conduction.
 - (c) Râles—Frictions.
- (b) Heart—
 1. Inspection: Precordial pulsation, precordial bulging, apex beat.
 2. Palpation: Thrills, apex beat, type of pulsation or heart action.
 3. Percussion: Outline of heart, outline of large vessels at base.
 4. Auscultation: Type of heart beat, rate and rhythm; murmurs, description and transmission, adventitious sounds.

Description of 1st and 2nd sound at each heart area—mitral, aortic, pulmonary, tricuspid.
- (h) Spine: Inspection.
 1. Asymmetry—
 - (a) Postural: Have patient fold arms and bend forward. If asymmetry disappears, it is due to posture.
 - (b) Real is a permanent anatomical change.

2. Attitude. In certain cases such as sacro-iliae, subluxation, this is almost typical.
3. Rigidity of muscles.
4. Spinal curves.
 - (a) Disappearance of normal.
 - (b) Exaggeration of normal.
 - (c) Fixation.
 - (d) Lateral curve.
 - (e) Rotation.

5. Movements.

Flexion of the spine to the right and to the left, with the patient's feet together, the knees and hips straight, should be requested; and movements of the spine noted and eliminate the difference in the curve described by the muscle, which is sometimes misleading. This is especially true in patients with superficial fat and in men with very large back muscles.

In forward flexion, note disappearance of the lumbar curve by either posterior convexity or flattening, noting any segment of limitation in any of these motions, and the point at which the curve begins; whether it is above the fifth lumbar or at the junction of the lumbar and the sacral vertebræ.

ROTATION OF THE SPINE

Patient's hips should be held in a fixed position and he should be requested to turn to his fullest ability to the right and then to the left, looking over one shoulder and then over the other. Normally he should be able to rotate 90 degrees in either direction.

Hyperextension of the spine should be requested and mental note made of the amount of lumbar curve possible. During this examination the patient should be requested to put one finger on any spot of pain elicited by any of these motions and this spot should be marked with an indelible pencil.

The patient is then requested to sit on the examining table with his legs straight out on the table and his knees in full extension, hips at right angles. This corresponds, of course, to a position of right angle flexion of the spine; and if he can assume this position, it is the position of full forward flexion, and can be checked with the amount of forward flexion that the patient demonstrates in standing. The only

difference being that there is less weight or strain put on the lumbar muscles. The position of the spine would be the same as in right angle flexion.

The patient is then asked to lie flat on the table and it is noted whether his legs come into full line with his body on a level with the table, as opposed to a stooped position which he may assume when he stands up. In sacro-iliac subluxation it is just as impossible for a patient to straighten his leg while lying down as while standing up; and hyperextension of the leg is just as painful to the patient standing or lying down.

While he is on the examining table, note is made of the amount of lumbar curve, by placing the hand beneath the curve while the legs are in full extension and whether this curve disappears when the thighs are brought up to right angle flexion. He is then asked to turn face down on the table and attention of the examiner is drawn to the ease with which the patient turns. This involves the use of the muscles of rotation of the spine and hips, and is an extremely difficult act to perform in patients with actual inflammatory condition in the lumbar and lumbo-sacral region.

Following this, the patient's elbows should be held at the side and he should be requested to stand in a normal posture while the examiner places his arms under the patient's elbows and lifts the body weight of the patient by a straight upward lift, without hyperextension. This, of course, removes the weight from any inflamed areas and should give relief and not pain. We know of no condition existing in the spine which should give pain on a straight upward lift of this type; and if the patient complains of pain it is reasonably sure that he is at least exaggerating his symptoms.

Palpation.—A manual examination of the entire spine from the base of the skull to the end of the coccyx should be made, marking on first examination the tender spots elicited.

This should include pressure over the sacro-iliac joints and over the exits of the sciatic nerve at the sciatic notch. All points of tenderness should be carefully marked with a skin pencil. Rigidity of muscles should be noted.

There is a method of examination introduced by Doctor Freiberg, of Cincinnati, in which a palpator of measured resistance is used. A rubber button the size of the end of a man's thumb on a rod which in

turn is connected with a spring scale. In this way the amount of pressure being used on the spine can be estimated and checked up. This is one of the best malingering tests of which we have knowledge. The tender areas of the spine are gone over with the patient's attention concentrated on the examination of the back, and it is noted at what pressure weight he complains of pain. A second examination is made a few minutes later, after the patient's attention has been somewhat distracted and a check up made to see whether again the same amount of pressure is necessary. If two pounds of pressure are used one time and eight ounces another to elicit pain, and this repeatedly varies to a considerable degree, and at the same time the points vary in location, it can be estimated that the patient does not have the amount of pain and tenderness which he states he has. Again, if the points vary it can certainly be said that they are not tender points due to any inflammation or organic trouble, inasmuch as tenderness due to inflammatory process does not change its location in the course of an examination.

Measurements.—McKendrick in "Back Injuries" gives a useful measurement of forward flexion. A tape measure is used and the commencement is laid over the first dorsal spine, the second spot located is the last dorsal spine which is usually twelve inches below the first; the third is the spine of the fourth lumbar and the final is between the posterior superior spines.

During the first measurement the patient should be in the normal standing position and the second measurement is made when he bends forward as far as he can, the tape being held at the first dorsal vertebra. It is found that there is no increase in distance between the first and twelfth dorsal spine, but that the distance between the last dorsal spine and the fourth lumbar has increased on the average about one and one-half inches, and the distance between the fourth lumbar spine and the posterior superior spines has increased about one inch.

Percussion.—Patient should then be requested to fold his arms in front of him and stoop forward to an angle of about 45 degrees. With the examiner's left hand flat on the base of the cervical spine at its junction with the dorsal, the back of the left hand should be struck a fairly heavy blow with the clenched right hand, giving a sharp jar to the patient's body and he should be requested to state, if there is any pain elicited, where this pain occurs. The patient's position, with

the blow directed forward and downward will naturally put sudden strain on especially the lower lumbar and sacral region. While the blow is not struck from this point, if there is actual tenderness at the attachments of the lumbar muscles or in the ligamentous support of the lumbo-sacro-iliac region, this blow will bring it out. Patient should then stand erect and without calling his attention to what is being done, a conversation being kept up continually, the examiner's hand should be placed on the shoulders of the patient and gradual pressure exerted. If there is an inflammatory process in any of these articulations along the spine or a breaking down or inflammatory process in the bodies below the cervical region, pain should be elicited at the points involved in the inflammatory process.

(i) Abdomen:

Inspection:	{	a Symmetry	{	abdominal thoracic
		b Shape		
		c Contour, movements, resp.		
		d Peristalsis		
		e Eruptions		
Palpation:	{	For abdominal organs	{	
		For tumor masses		
		For tender areas		
		For enlarged glands		
		For rigid muscles		
Percussion:	{	For outline of organs	{	
		For dull, flat and tympanitic areas		
Auscultation:		Peristalsis		

(j) Extremities:

Describe in general—eruptions, muscle spasm, tender areas, glandular enlargements, muscle atrophy.

(k) Nervous System: Reflexes—Biceps

Triceps

Patellar

Achilles

Babinski

Ankle Clonus

Romberg.

- (1) Laboratory Reports—Urine
Blood
Sputum
Feces
Blood Culture
- (m) X-ray Report

DIFFERENTIAL DIAGNOSIS—CERVICAL PAIN

Injuries.—It is surprising to what extent an injury may involve the cervical spine without producing any other symptoms than a continuous pain and a stiff neck. This is well shown in a previous case quoted in *INTERNATIONAL CLINICS* on page 192, vol. ii, series 31, fracture dislocation of the fifth cervical vertebra. These severe injuries are easily diagnosed by the X-ray, but the minor injuries, especially if followed by an arthritis, are extremely hard to diagnose, and the diagnosis must often be made by elimination. In cases like this, with a history of an accident, with pain and rigidity of the neck immediately following with definitely localized tenderness to one or more of the cervical vertebræ with no positive X-ray findings, a diagnosis should be made of strain of the muscles or ligaments of this region.

Torticollis.—There are various forms of torticollis, the acute form, or the ordinary stiff neck, is a common condition, of sudden onset with the affected muscle sensitive to pressure. In this the pain is acute and it usually clears up in a short time.

Congenital torticollis is usually not accompanied by pain.

There is an infectious torticollis due to infections in the cervical glands, otitis media, tonsilitis, pharyngitis or other infections in the cervical and submaxillary region. In this, the onset is usually sudden, the source of infection is usually easy to be found; but this condition may last for a considerable period and the infection may be difficult to find so that in all cases where there is an intense pain in the muscles, the subject of focal infection must be thoroughly considered. Torticollis is caused, of course, by a contraction of the muscles supplied by the spinal accessory nerve and if the tense muscles are relaxed by inclining the head toward them, motions in the spine itself will be found to be free and painless.

Cervical Potts' Disease.—Potts' disease in the later stages is diagnosed by X-ray, but in the earlier stages, the röntgenological

examination is of little value because the symptoms are usually severe before there are destructive changes in the bone. In these early cases, the restriction of motion due to muscular contraction is the most important sign. This restriction of motion in the upper cervical region is more a restriction of flexion and extension of the head on the spine, and in Potts' disease of the upper cervical region the attitude of the patient is somewhat characteristic, as the neck is fixed. There may be a slight bulging or infiltration about the diseased vertebræ. There may be a fluctuating mass in the sub-occipital region. Flexion of the head on the spine is limited; also rotation of the head. The inspection of the posterior wall of the pharynx may show a bulging beginning abscess formation. If the deformity is present, the diagnosis can be made by X-ray.

Retro-pharyngeal Abscess.—This may cause chronic cervical backache. It may be due to Potts' disease; may be one of the sequelæ of a contagious disease, or pharyngitis. The diagnosis is made on the fever, dysphagia, enlarged cervical glands and by inspection and palpation of the posterior wall of the pharynx.

Anatomical Deformities.—It has been the experience of the authors that anatomical deformities usually occasion no pain until there has been some slight strain. The most common deformity in the cervical region is the presence of a cervical rib. Usually this is bilateral, but may be unilateral. In these cases, after some strain serious symptoms may be caused due to the pressure on nerves and blood-vessels. In the upper cervical region the atlas is subject to variation especially. The posterior arch may be incompletely ossified or it may be assimilated into the occipital bone. The odontoid of the axis may be detached and the atlas and axis may be fused. These upper anatomical peculiarities seldom cause difficulty.

PAIN IN THE THORACIC REGION

Injuries.—Again, it is to be remarked that serious injuries of the thoracic vertebræ may be noted with the patient complaining of pain in the thoracic region or pain in the lumbar region. A case quoted on page 1, vol. iii, series 30, of *INTERNATIONAL CLINICS*, illustrates this injury.

Round Shoulders.—This is a postural deformity which may cause chronic backache in the thoracic region due to the position and the

muscle weakness. In this condition the deformity is present, has practically no localized pain or tenderness; a history of slow onset with usually some occupational condition. There is practically no rigidity.

Thoracic Potts' Disease.—This disease is often slow in its onset. There is usually evening temperature rise, a definitely localized pain and tenderness. There may be a cough. The X-ray examination shows this condition in its later stages. It is to be noted that the normal curve of the back in this region is with the convexity backward and a deformity for this reason may be hard to detect. Potts' disease may cause pain only after aggravation by an injury. This condition is shown in a case previously quoted in *INTERNATIONAL CLINICS* on page 36, vol. i, series 31.

Mediastinal growths or glandular enlargements may cause severe and persistent pain in the back. The shadows of the X-ray picture cast by the enlarged glands are usually decisive in this condition.

Chest affections such as pleurisy, pneumonia, empyema and pulmonary tuberculosis may cause pain in the thoracic region which may be accompanied by a lateral deviation or a rigidity of the spine. In these conditions a careful physical examination of the chest will bring out the real cause of the pain.

Atheroma or aneurysm of the thoracic or abdominal aorta may cause pain along the spinal column in the thoracic region. In aneurism the X-ray and the fluoroscopic examinations will show the shadow of even a small sack and there are usually other characteristic physical findings present.

Affections of the heart such as pericarditis will occasionally cause pain in the thoracic spine, but again the characteristic symptoms of the condition are present on the physical examination that includes the heart.

Gastric Ulcer.—In many cases of gastric ulcer there is a well-marked pain at the level of the tenth dorsal vertebra, but these attacks of pain are frequently induced by taking food and usually grow at intervals; and there is an epigastric tenderness likewise and an examination of the stomach contents will show an abnormal acidity.

PAIN IN THE LUMBAR SPINE

Injuries in the Dorso-lumbar Region.—As previously stated, the fracture dislocation is the most common of the injuries, which means that the twelfth dorsal vertebra is dislocated forward on the first lum-

bar. Oftentimes the pain in this injury is over the whole of the lumbar region, due to the strain on the erector spinæ muscle and holding the spine rigid.

Fractures of the transverse processes of the lumbar vertebræ are fairly common and cause more or less chronic pain, which is aggravated by motion. The history of the injury and the X-ray examination give the diagnosis in all except strains of the ligaments and muscles; and it is in this region that the pressure apparatus of Doctor Freiberg is especially valuable in diagnosing these conditions.

Lumbago is a painful affection of the lumbar muscles, usually following cold and exposure, and the pain may be dull ache, or sharp and severe. Pressure on the affected part usually gives relief. It is usually a transient affection. Focal infections usually play an important part in the etiology.

Potts' Disease of the Lumbar Region.—The stiffness of the spine which makes bending or turning of the body difficult is one of the first symptoms. The early stage of the disease is characterized in lordosis or over-erectness. Later there may be a characteristic deformity. The rigidity of the lumbar spine may be tested by hyperextension of the legs with the patient on his abdomen. Psoas contraction may be tested by placing the patient on his back with the pelvis at the edge of the table.

Tuberculosis or other conditions of the hip may give a reflex pain in the lumbar muscles. Diagnosis is made by examination of the movement of this joint.

Kidney Affections.—In unilateral pain in the lumbar region that gives tenderness at some distance to the site of the vertebra, kidney affections may be suspected. A case that we cite further on in this issue will illustrate this condition. The diagnosis of this is made, of course, by the urine examination, by catheterization of the ureters and by the X-ray.

Pelvic abscess may give a chronic and persistent pain in the lumbar region. These abscesses may be perinephritic, from appendicitis, tubal infections or from Potts' disease, etc. In these conditions there is usually a temperature rise, pain in the lower abdomen and a palpable mass to be felt by rectal or vaginal examination.

Visceroptosis often causes a chronic pain in this region with a dragging sensation. Visceroptosis usually occurs due to the loss of

the normal support of the abdominal wall from repeated pregnancies. With a chronic backache, a relaxed abdominal wall would lead one to further examine the gastro-intestinal tract by means of the fluoroscope and röntgenological examination after a bismuth meal.

Nephroptosis is another of the same sort of conditions that may cause a backache, although in this nephroptosis there is often found severe paroxysms of pain.

Constipation and intestinal intoxication are causes of backache which should not be forgotten and we had in this Clinic a man who had a chronic lumbar backache due to this condition; this backache had persisted for several years and he had been treated for many conditions without relief of the backache. Indican in the urine is one of the diagnostic symptoms that we have found in intestinal intoxication in these conditions.

Spondylolisthesis.—This is a deformity in which the lower lumbar vertebra is displaced forward and downward. There is pain and weakness in the lower lumbar region and there may be an apparent deformity in this region of a sharp forward inclination of the spine just above the sacrum. The X-ray will make the diagnosis.

Congenital Anomalies.—The anatomical peculiarities in the lumbar region have been somewhat taken up under the practical anatomy. Fig. 12 in vol. ii, series 30, page 21, shows a low lying fifth lumbar with a long lateral process, which is the X-ray of a case quoted on page 28 of the same issue. If any of these anatomical peculiarities are found on X-ray and the pain centers over the fifth lumbar vertebra, a diagnosis may be made that the pain is due to some strain of the ligaments in this region or to a rupture of these ligaments allowing the abnormally long transverse processes to impinge on the ilia or a nerve. Other congenital anomalies in this region are shown in Figs. 4, 5, 6, 7.

Bladder diseases or inflammations will also cause pain in the lower lumbar region, but in these conditions there will, in addition to the lumbar pain, be pain and tenderness in the suprapubic region and abnormal urinary findings.

Prostatic hypertrophy or inflammation often gives chronic backache, but in addition, there will be a history of frequent and painful urination, and rectal palpation will disclose the prostatic mass.

Ovarian, tubal, uterine and perineal affections in women will give

FIG. 4.



Congenital anomaly in dorso-lumbar region. Last rib on left has no counterpart on right. There is a wedged shape half portion of vert on left at first lumbar region.
(X-ray by Dr. H. E. Potter.)

FIG. 5.



X-ray by Dr. H. E. Potter shows lateral view of Fig. 4.

FIG. 6.



X-Ray by Dr. H. E. Potter showing incomplete closure of lamina fifth lumbar vertebra and upper sacrum which amounts almost to an opening sufficiently large for the development of a spina bifida.

FIG. 7.



Congenital defect in transverse process of third lumbar vertebra.
(X-ray by Dr. H. E. Potter.)

a chronic lumbar backache, and no diagnosis of the causes of lumbar backache in women is complete unless a complete examination of the pelvic viscera is made.

PAIN IN THE SACRO-ILIAC JOINTS

The diagnosis of these conditions has been considered on page 1, vol. iv, series 30. It is to be remembered that not all pain and localized tenderness in these joints is due to trauma, but that such infections as gonorrhea and tuberculosis may involve this joint. We had one case in our Clinic in which after a second attack of gonorrheal urethritis, a man developed an acute gonorrheal arthritis in the left sacro-iliac joint and the right knee and right ankle. In the infections of this joint there will usually be marked pain and tenderness on lateral pressure on the ilia, which is not marked in strains. The other characteristic symptoms of these infections will differentiate them from the injuries to this joint.

In subluxations of this joint the condition can rarely be demonstrated by an X-ray picture. At times an arthritis following injury is plainly shown in the X-ray.

PAIN IN THE SACRAL REGION

Injury.—Fractures of the sacrum cause persistent pain and tenderness in this region, as is illustrated by the case quoted on page 191, vol. ii, series 31.

Malingering.—One of the ways that we have found useful in detecting malingerers is on complaint of pain and tenderness over the sacrum on spinal motions, when there are no X-ray findings or pelvic diseases that would cause such pain, as there are no muscles or ligaments attached to the posterior surface of the sacrum.

Rectal diseases such as fissure or carcinoma may cause sacral pain, and no persistent sacral pain can be diagnosed without a thorough proctoscopic examination.

Pelvic viscera displacement and diseases will also cause sacral pain, and we believe that it cannot be too often emphasized that in all cases of backache in women there must be a thorough pelvic examination.

Coccygodynia.—Pain and tenderness in the region of the coccyx may be due to injury to the coccyx, diseases of the pelvic organs or tissues such as hemorrhoids, rectal and anal fissures, fistula. It may

follow childbirth and without injury it is usually seen in hysterical and neurasthenic women.

GENERAL CAUSES OF BACKACHE

Lateral Curvature of the Spine.—In these cases the deformity is usually preceded or accompanied by pain and this is usually more of a pain of fatigue than it is acute. The diagnosis is to be made upon the deformity and the history of the case, as lateral curvature is due to certain predisposing causes such as shortening of one of the limbs; secondary to a paralysis such as anterior poliomyelitis; or following a disease of the chest, such as empyema; due to occupation or congenital.

Syphilis may affect the bones of the spine in the tertiary or inherited stages, but it is an extremely rare condition; and the diagnosis can only be made with the Wassermann or the previous history.

Sarcoma or carcinoma gives more severe and persistent pain than any of the other conditions considered and cachexia soon appears and the disease rapidly goes on to a fatal termination.

Infectious Diseases.—Typhoid, scarlet fever and other infectious diseases may cause pain, weakness and stiffness of the back, due to an infection of the periosteum and articulations of the vertebræ. The diagnosis is usually made by the history of the condition following an infectious disease.

Spondylitis Deformans (Spinal osteoarthritis).—This is a chronic progressing disease terminating in ankylosis and deformity. It is commonly seen in male laborers. Oftentimes there is an acute onset possibly following some minor strain or traumatism. When the patient is X-rayed there is found to be an astonishing amount of ankylosis; we had a case in which a man aged forty, after lifting, complained of sudden and severe pain in the lumbar region. He never had pain previously and had always worked hard. Yet the X-ray indicated a condition that would indicate that the process had been going forward for a long time.

Rhachitic Spine.—This deformity is a rounded curvature of the lower part of the spine and the diagnosis of the condition can be made on the history and the indication of the condition in other parts of the body.

Osteomyelitis of the spine (extremely rare condition) is usually associated with severe local pain, fever and constitutional symptoms.

PYELITIS—UNILATERAL, CAUSED OR AGGRAVATED BY TRAUMA

D. B., male, age 38, married, one child.

Family History.—Father and mother killed in the war. His wife is living and well. She has had one miscarriage, one child died at the age of four months and one is living at the age of 26 months.

Previous Medical History.—Shows he has never been sick before, but in May, 1920, a five-hundred-pound weight hit him in the left lumbar region, and he was off from work during May and June and part of July, and during July and August of 1920, he had great pain in the left lumbar region and difficulty in passing urine, July 19, 1921, while lifting a heavy weight, he had sudden pain in his left lower lumbar region. This pain was so bad that he could not straighten up. He was home for three days in bed, but went to work again on July 22 and 23. He was first examined in our Clinic on July 26.

Present Complaint.—Pain in the left lumbar region, weakness and loss of weight.

Examination in our Clinic showed all reflexes normal; no glandular enlargements. Teeth and tonsils negative. Lungs negative; after a careful examination heart was found to be negative.

In the spine he had pain and tenderness over the lower lumbar spine, over both lumbar regions and all motions of the spine were limited. For the above reasons he was X-rayed and there was no evidence in the lumbar spine or sacro-iliac regions of fracture dislocation or disease.

After about a week of radiant heat treatment, the pain in the back entirely cleared up except in the left lumbar region.

Urine showed a trace of albumin with blood cells and pus cells, and the colon bacillus and staphylococcus; a guinea pig inoculation showed no tubercle bacilli although this was tried on two guinea pigs. Catheterization of the ureters showed pus coming from the left kidney.

Diagnosis.—Left pyelitis, mixed infection.

COMMENTS

Causes.—Causes of infection in the kidney as elsewhere in the body may be stated as a lowered resistance of the tissue and an organism capable of infecting the kidney, which usually comes from a focus elsewhere in the body. Most of the infections are probably hematogenous. The pelvis of the kidney is predisposed to infection or inflammation by irritation and congestion. Among the irritants are mentioned drugs, highly concentrated urine, renal calculi. Clots may cause congestion as may stone in the kidney, ureter or bladder, or a pressure upon the ureter from without by abdominal tumor or kinking of the ureter in a movable kidney, by ureteral stricture or by a prostatic enlargement.

In this case the question of trauma was disputed as a cause and a somewhat careful study of the literature was made. Keyes says

that the surgical infections of the kidney are microbic in origin and may be caused by any pyogenic bacteria. He gives in his predisposing causes that bacteria are always present. Every attack of constipation sends millions of colon bacilli through the kidneys and from every infected wound a sufficient number of staphylococci and streptococci doubtlessly enter the circulation and pass through the kidneys to cause suppuration ten times over in these organs, if only they are vulnerable, but they are not. Unless there is some trauma, irritant or congestion the bacteria are passed off without so much as multiplying in the urine. Martin, Thomas and Moorehead, in their "Genito-urinary Surgery" say the most frequent predisposing and exciting causes of pyelitis are (a) the infectious diseases; (b) traumatism, a rare but undoubted cause; (c) et cetera. Cabot, in his "Modern Urology," vol. ii, under accessory causes of kidney infection, says under trauma, that "We may include in traumata that occasion renal infection not only such open wounds that at once introduce bacteria and harrow the soil for their reception, not only the sub-parietal contusions and lacerations that harrow the soil to await the seed; but also the trauma of slight bruises and wrenches, sufficient to contuse the kidney and yet evoke no symptoms, as well as of renal stone which forms at once a source of irritation and nidus for infection."

Organisms.—In this case the organisms found were those that are usually found, the colon bacilli and the pus organisms, and while from his loss of weight and blood cells in the urine tuberculosis was at first suspected, tubercle bacilli were never found either by staining or by culture or by guinea-pig inoculation.

Symptoms.—This man had the typical symptoms of pyelitis; pain in the left lumbar region which was severe and he had acute exacerbations. This pain was increased by motion.

The urine was acid, as is usual, showed a trace of albumin and contained blood and pus cells. The diagnosis of a unilateral infection was made upon the catheterization of the ureters.

Several times during the course of our treatment, this man had chills and sweats with exacerbations of temperature. He ran a continuous elevation of temperature at night.

Treatment.—Patient was given an autogenous vaccine so standardized that each three mms. contained 75 million of the organisms, given hypodermically in initial dose of 3 mms. It was so given that

FIG. 8.



Results of breaking up adhesions in stiff joints, posterior dislocations, fracture of the neck and of the shaft of the femur.

this injection produced an aerola about the site of the injection, which shows itself in about twelve hours. The second injection was given the day after this reaction had disappeared. When the dose given produced a reaction, the dose was not increased, but if it did not cause a reaction, the dose was increased and the dose repeated that produced a reaction until it no longer did so. In our case the average time for the dose was about three days and the largest dose given was about eight mms. The man under this autogenous vaccine treatment and urotropin in large doses has shown a steady improvement and has gained ten pounds in weight and is now working, but not at as heavy work as he was before. If this had not been effectual, pelvic lavage with a weak solution of some of the silver salts was to have been tried, but so far this has not been necessary.

STIFF JOINTS

The following case illustrates in a startling manner that the subject of stiffness of joints is not only not understood by the members of our own profession, but it is still less understood by those professions to which many people give great credit and ascribe great power in the treatment of such conditions; namely, the osteopaths.

M.K. Age 47, occupation farmer; married.

Family history is negative.

Previous personal history is negative except that twenty years ago he had "white swelling" in the left knee joint.

History of Present Illness.— About one and a half years ago the patient had "rheumatism" in the right hip joint. This was extremely painful and lasted for some weeks. During the time it was painful he flexed and adducted his thigh and hip; when the pain disappeared the joint was stiff and was held in this position. About three months after the original attack of pain, when the pain had entirely subsided, he went to an osteopath who placed him on the table and by several manipulations forcibly hyperextended his leg from its position of flexion and adduction. After this he had considerable pain and could not hold his leg in the extended position and it again became fixed in the flexed and adducted position. Some weeks later he went to his family doctor, who called in a local surgeon and under an anæsthetic they forcibly "broke up the adhesions." Since that time he has been in intense pain; unable to leave his bed. His leg has been fixed in flexion to about 75 degrees and adducted. His knee flexed to an angle of 90 degrees and any motion of his right leg has caused him intense pain and marked muscle spasm.

X-ray of this hip, as shown in Fig. 8, shows a most remarkable state of affairs and shows that the adhesions were certainly broken up.

There is a posterior dislocation of the head of the femur and fractures of the neck and of the shaft. This case illustrates exceedingly well how strong some of the adhesions are and shows that at times the retracted soft tissues may be so resistive that the bone may be broken instead of the adhesions.

In view of the many cases that have come to our notice it is thought well here to give in detail the treatment of stiff joints as it is carried out in our clinic.

TREATMENT OF STIFF JOINTS

Treatment of stiff joints must be divided into two classes—preventive and curative.

Preventive treatment of stiff joints consists in early massage and motion in the injured joints, and in our Clinic we have found that one of the greatest aids to the prevention of stiffness in the joint motion is the galvanic current with clay electrodes. The technic of massage can only be learned by experience and practice on patients and should be given only by one who has had considerable special training. This technic is given in many books and in most cities there are trained masseurs. Massage and joint motion should only be given under specific direction or supervision of the attending surgeon.

For the prevention and treatment of stiff joints we have found the clay ionization very successful, and it is being used by another industrial surgeon in this city with great success. The clay is mixed with water and glycerin and kneaded until smooth. The clay is applied to the joint or part to be treated, completely covering the joint. The negative terminal of the galvanic current is placed in the clay, usually using a bifurcated cord. The indifferent electrode should be thoroughly wet in hot water and rubbed with soap inasmuch as the contact is much improved when it is wet. The current is regulated to suit the tolerance of the patient. This treatment should be carried out daily.

1. *Mild Types*.—In the treatment of sprains the torn ligaments should be relaxed at all times so that it is extremely necessary to get an accurate history and make a diagnosis of the ligaments that are torn, so that the joint should be fixed in such position that the ligaments are relaxed.

Manipulations and movements of the joints should be delayed

in these cases for a few days, say about the third day; and when manipulations are started they should be in such a direction that the torn ligaments are not strained.

Exudation and effusion of blood and serum into the joint is prevented by either adhesive strapping or a compression bandage over cotton.

Massage should be started in a few days and should be aimed at the absorption of the hæmatoma and the stimulation of the circulation. This is accomplished by local kneading and friction, with general massage of the whole limb. What should not be done in strains is immobilization.

In dislocations the first question is the reduction of the dislocation and mobility should be maintained from the onset, but the joint motion should always be carried on by the surgeon himself, remembering exactly how the dislocation occurred and not putting any strain upon the torn ligaments. The after-treatment of dislocations in our clinic is by massage, heat and radio clay is again used to prevent the formation of adhesions.

Stiffness due to prolonged immobilization is, of course, best treated not as a curative measure, but in the preventive treatment this has been emphasized in previous issues of our clinics under the subject of fractures.

Stiffness due to contusion of joint cartilage—this condition we have seen most often in industrial cases in the knee. These should be treated by rest for about two weeks and then the joint should be gradually taken through its range of motion.

2. *Severe Types of Stiff Joints—Bony Obstruction.*—Stiffness in joints due to bony obstruction is easily diagnosed by the actual feel of the motion when it apparently ends by contact of bone against bone, and again, the X-ray will clearly show just where the blockage occurs. In industry when we have a bony blocking, the following should be thought of in the order of their importance: (a) Excessive callus formation; (b) deformity of a malunited fracture; (c) any reduced dislocation.

The treatment of this condition is the treatment of the cause and it is usually operative.

Contraction of scar tissue causes stiff joints and several of our previous clinics have illustrated this type of stiff joint. If the scar

is situated in certain places, such as the back of the knee or in front of the elbow, there is great tendency for the scar to go on contracting until the member is fixed in a perfectly useless position. This condition in the back of the knee is seen in a case previously quoted in vol. ii, series 31, p.179, entitled "A Case of Bad Judgment." The stiffness of a joint may be solely due to the contracture of a scar tissue, involving the skin alone, or this scar tissue may be adherent to the muscles or even to the bone. Scar tissue is extremely strong and unelastic. The scar tissue, if it is loosened by repeated minor efforts after each of these efforts, the scar is damaged, the damage is repaired by new scar tissue, and so the scar is built up rather than diminished. The scars can be loosened by either constant tension, which slowly stretches them, by massage and manipulation, or by voluntary muscular effort. Again, in our Clinic, we use galvanic current with the clay electrodes to assist in this treatment of adherent scars and cicatricial contractures, the length of the treatment being from fifteen to forty minutes, and is given only in scars that are well organized.

If the scar is greatly contracted, it is to be remembered that the treatment by massage and joint motion without the aid of splints to constantly stretch the scar, is usually unprofitable work, whereas the stretching of this unelastic fibrous tissue is slow and must be carried on throughout the twenty-four hours.

Oftentimes the above measures fail and operative treatment of scars is necessary, especially if they are adherent to muscle or to bone, or are painful. In operative treatment of scars, if it is not possible to dissect sufficient skin flaps to eliminate the scar area, skin grafting or transplantation must be used.

3. *Severe Fibrous Ankylosis.*—This usually follows a very prolonged immobilization after fractures, or is due to an attack of arthritis, as seen in the illustrative case given in the previous article, or to suppuration about a joint. This is the class of cases in which we usually see the so-called breaking up of adhesions under an anæsthetic, and it has been our experience that more harm than good has been done by this method of treatment and this illustrative case brings to our mind the great harm that can be done by forcible manipulation under an anæsthetic. We believe that repeated forcible manipulations favor and hasten bony ankylosis. The joint is a

very delicate thing and forcible manipulations under an anæsthetic is a severe injury to a joint. The joint responds to this injury as it does to any other injury and there is formed a hæmatoma within the joint which rapidly goes on to organization and the formation of more fibrous adhesions.

Forcible manipulation in the hands of the experienced surgeon at times is very useful. The best rule on breaking down of adhesions is given in vol. i of Jones "Orthopedic Surgery of Injuries," as follows:

1. Before breaking down adhesions, exclude the presence of active arthritis.

2. In breaking down adhesions and stiffness following traumatic arthritis we must proceed very slowly and if inflammatory reaction supervenes, the manipulations should not be repeated.

3. Joints stiff following septic conditions within or immediately without a joint should not be submitted to forcible manipulation.

4. Adhesions are best broken down under complete anæsthesia and the joint should be put through its complete range of movement; otherwise a recurrence of stiffness may be expected. (We would like to add under this rule that this should only be done in certain selected cases and by an experienced surgeon.)

5. Sudden, jerking movements must be avoided because they are inefficient, cause unnecessary trauma and may break the bone instead of the fibrous band.

There are many surgeons who seem to believe that if one manipulation under an anæsthetic does not help that this should be repeated until the joint is free. We have seen one case of an elbow joint that has been manipulated every two weeks for the last six months, and the only gain that we have been able to observe is the gain in the size of the elbow joint, which has increased in size after each manipulation.

Bony Ankylosis.—This is a true ankylosis in which bone is united to bone without the intervention of other tissue and is usually the result of suppuration in the joint. If ankylosis is feared, due to suppuration in the joint, the surgeon should know the position of election for the various important joints for this point.

Shoulder joints should be ankylosed in a position so that the arm abducted to about sixty degrees from the side; the elbow should be in such position that the hand is toward the face. The arm should be placed in this position so that the hand can easily reach the face.

It should be remembered in septic shoulder joints, the arm should not be bound to the side, as is often done.

The elbow joint is usually fixed at a right angle which enables a patient to go through most of the necessary motions, including eating and combing his hair. A stiff elbow in extension makes practically a useless arm.

Hip joint ankylosis, the position of election is slight abduction flexion and outward rotation.

In the knee, position of election is in the position of about 135 degrees of extension.

The ankle is best ankylosed in a position of slight dorsal flexion beyond a right angle.

In any discussion of stiff joints it is to be remembered that in tubercular joints, rest and ankylosis are desired.

Operative Treatment of Ankylosed Joints.—In the operative treatment of ankylosed joints there has been considerable discussion of the advantages and disadvantages of arthroplasty over excision in the elbow joint, and we believe that the following case is an excellent argument for arthroplasty of this joint.

B.A., age 37, white, male, cigar merchant.

Admitted 6-22-20—Discharged 10-9-20.

Chief Complaint.—Stiffness of the right elbow; stiffness of the left elbow, with some pain in this elbow and tenderness and heat.

History of Present Illness.—About twenty years ago the right arm became stiff and painful; there was a fluctuating mass over the region of the elbow which, when incised, was found to contain pus and subsequently this elbow, through loss of function, adhesions and because of pain, was not used, and as a result ankylosed. During the past year the elbow on the left side has become stiffened and ankylosed. This came on gradually. Patient was able to feed himself until about two or three weeks ago when the left arm became so that he could not carry an ordinary length fork to his mouth.

There is some pain in the left elbow, but not enough to keep the patient awake; pain is not constant. The right leg at the knee became stiff twenty years ago, at the same time the right arm became stiff.

Past Medical History.—Patient states that he had syphilis and gonorrhea following which the right arm and right knee were involved. The right eye also was involved in a Neisserian infection and at the present time is blind. There was a small ulcer formed at the same time under the lid of the left eye, which healed after being lanced, leaving no permanent injury. Patient had pneumonia three years ago, erysipelas six years ago.

Family History.—Father died of cancer; otherwise negative. Wife is living and well.

Physical Examination.—Patient is a fairly well developed male, blood-pres-

sure 128-84. The right eye is blind. Pupils react to light and to accommodation. Teeth are in poor shape, pyorrhea being in evidence. Tongue is coated. The ears are negative. Nose is negative. Neck is short and of heavy musculature. There is no pulsation of the large vessels.

Chest.—Emphysematous in type; expansion is good and equal; no râles. Heart apex beat is normal; muscle tone is of good quality; no murmurs.

Abdomen is negative; no areas of tenderness or tumor masses.

Extremities.—Examination shows the right elbow fixed in about 15 degrees of flexion and the forearm in semi-pronation. The rigidity is that of a solid, bony ankylosis; patient states that the arm has been in this condition for the last eighteen years following an infection, Neisserian in origin.

The X-ray shows a firm, bony ankylosis between the radius and ulna and between the ulna and humerus, and also between the radius and humerus.

There are scars of what apparently were drainage wounds long healed, over the posterior surface of the right elbow over both the inner and outer condyles. These scars are firmly adherent to the bone beneath.

The left elbow shows about 10 degrees of voluntary motion and not over 15 degrees of passive motion. Pronation and supination of the forearm are normal. Patient is unable to feed himself except by the use of a long handled fork and spoon; the handles of which are made in three sections which he carries in a special pocket; these sections he screws together, making long handled instruments with which he may feed himself by the wrist action alone.

The X-ray shows no bony ankylosis, but considerable irregularity in the bone outlines, indicating a thickening around attachments of the ligaments and an arthritis of long standing. There is some heat and swelling in this joint and much tenderness.

The right knee is ankylosed firmly by union between the tibia and femur and between the patella and femur. This has existed for eighteen years, the same length of time as the right elbow and is perfectly painless.

In view of the fact there is an acute flare-up in the left elbow, which has become more rigid within the last few months, present operative procedures are deemed inadvisable. Patient is to be treated by heat to the left elbow and massage; and exercises of the muscles controlling the right elbow are to be started immediately for the development of the muscles in this arm which control the right elbow.

NOTE: Aspiration of the fluid in the left elbow shows clear, serous fluid with a few pus cells, but no bacteria.

Laboratory.—Blood Analysis—Red cells 4,600,000; whites 15,500.

Urine—A trace of albumin; otherwise negative.

Wassermann—Alcoholic extract negative. Cholestrinized extract negative.

Smear from urethra following prostatic massage shows many pus cells and many extra cellular diplococci; few cells show intercellular diplococci.

Smear from urethra—no gram negative cocci present; few gram positive cocci present.

Examination of the mouth shows some inflammation of the mucous membrane and some calcareous deposits on the teeth and the presence of some decay.

Operation (8-5-20)—Right elbow.—Incision along the inner condyle of the elbow, flap reflected backward and ulnar nerve exposed and dissected free; held out of place by a smooth, ribbon retractor; a chisel to conform with the curve of the

condyle of the humerus was driven straight through at an angle corresponding with the carrying angle of the humerus toward the radial side; a smaller curved chisel was then driven between the internal condyle and the olecranon and the olecranon fossa gouged out. A considerable portion of bone, probably in all amounting to $\frac{3}{8}$ inch, was removed from between the ulna and the humerus in as near the form of the normal joint as possible. This being completed, wound was covered and a second incision made over the head of the radius, the head of the radius amputated at the neck and chiseled loose from the humerus above and removed. A channel was opened up in this way through and through; the forearm displaced backward angulated through the external wound; the future articulating surfaces of the ulna were then smoothed off carefully, a very substantial coronoid process and the olecranon being allowed to remain. These bones were then angulated back into the wound and the end of the humerus forced out into view and these surfaces smoothed off. The coronoid fossa and the olecranon fossa being gouged out so that there was no bone remaining between them. Over the end of the humerus was placed one large piece of Allison's membrane, a silver impregnated fascia, which had been previously moistened and softened by placing in sterile water. This membrane was sutured around the head of the humerus, fastened both in front and in back and sutured together at the sides, leaving the whole lower end of the humerus covered. The ulna was then placed in contact with the membrane, the elbow put in 90 degrees of flexion, and the lateral ligaments sutured with fine catgut and the skin closed with silk-worm gut.

Temperature 98, pulse 110.

The day following the operation temperature 100.6, pulse 108.

8-21-20— Temperature 101.8, pulse 120. There was some swelling in the elbow and some heat. Stitches were removed and a small quantity of purulent material escaped.

8-22-20— Temperature went down to 99.2.

8-23-20— Temperature went up again to 101. From that time on the temperature gradually came down to a normal line. A further spreading of the wound was made and a small guttapercha drain inserted.

8-26-20— Active motion started.

8-27-20— On this date temperature advanced to 100 with no swelling but considerable soreness in the elbow. The active and passive motions were persisted in.

The temperature gradually subsided, the drain removed and the wound closed entirely.

9-1-20— Temperature normal. Patient encouraged to use his elbow and has 45 degrees of voluntary motion.

9-25-20— Patient has developed 60 degrees of motion actively; the triceps and biceps have improved much in strength.

10-9-20— Patient is discharged with 90 degrees of voluntary motion.

Report One Year After Operation.—Patient was seen in Galesburg, an examination of the elbow showed firm lateral stability; no lateral bend at all on full extension of the arm. Extension of the arm was normal and flexion 115 degrees.

CARCINOMA OF THE TRANSVERSE COLON CAUSED BY INJURY

The following case of carcinoma of the transverse colon, confirmed by microscopical examination after operation, brings up many medico-

legal questions, as compensation was demanded under the Workmen's Compensation Act, due to death being caused by carcinoma following an injury.

Trauma in relation to malignant tumors, that is, as an etiological factor, is one of the theories that is commonly advanced as a cause of carcinoma. The usual case in which trauma is given as a cause is that in which the irritant acts over long periods of time, such as cancer of the lip from smoking of pipes, cancer of the tongue from irritation of a broken tooth, cancer of the cheek due to chewing of buyo leaves, cancer of the skin that is seen in X-ray workers. Very few well-established cases are on record of a cancer that can be ascribed to a single trauma. Many women with cancer of the breast ascribe it to a blow, but usually the blow called attention to the cancer that was already present. The subject of a single trauma producing cancer is rather indefinite. It is fairly well agreed that sarcoma may arise in a bone after a fracture or injury to the bone. Here there is usually no discussion, as the tumor to come under this classification must arise at the site of injury. William B. Coley, in the *Annals of Surgery*, 1911, made an extensive study and collected all the literature of this subject up to that date. He says that he has only personally observed a few cases of intra-abdominal cancer, definitely associated with antecedent trauma. He quotes two cases as follows:

CASE VIII.—E. P. F., male, forty-four years of age. In December, 1898, in an accident on a railroad, patient was thrown violently against a steel tank, striking in the upper abdomen, causing marked ecchymosis, nausea, and pain, some vomiting of blood which lasted for two to three weeks. He continued to get worse, and in February, 1901, he was seen in consultation by Doctor Delatour. No tumor could be felt at that time. In May, 1901, a mass could be made out in the right upper abdomen. An exploratory operation was performed, and a large number of tumors of the mesenteric glands were discovered. The patient died of shock following the operation. Autopsy showed the mesenteric glands in the upper abdomen markedly enlarged, some being the size of a hen's egg. The pancreas was likewise involved by similar growths. Microscopical examination showed the growths to be sarcoma. At the first trial in court the jury disagreed and a settlement was affected before the second trial was called.

CASE IX.—Intra-abdominal cancer following trauma. Carcinoma of the ovary: Mrs. W., aged fifty-seven years. Family history good. In the early part of 1909 had such a severe fall upon the ice, that she felt as though her bladder had been ruptured. Three or four weeks later noticed a lump in the lower abdomen. She was operated upon by Doctor Wynkoop March 16, 1909; a large tumor was removed from the right ovary. Microscopic examination showed it to be malignant. July, three months later, a very extensive recurrence was found occupying the whole lower abdomen. This increased very rapidly in size. I saw patient in August, 1909, at which time the whole abdomen was filled with a large tumor apparently connected with the uterus. It had the appearance of carcinoma rather than sarcoma. Toxin treatment was tried for a number of weeks, but patient showed no improvement. Death occurred a few months later.

From his article and Segond's, it is believed that seven conditions should be fulfilled before a cancer can be considered to be definitely due to an injury. They are as follows:

1. The authenticity of the trauma.
2. Sufficient importance or severity of the trauma.
3. The integrity of the part prior to the injury.
4. Correspondence of the tumor to the exact site of the injury.
5. A date of appearance of the tumor not too remote from the time of the accident to be reasonably associated with it.
6. The continual presence of pathological manifestations, such as pain, swelling, hæmatoma, etc., at the site of injury, up to the time of the appearance of the tumor.
7. A histological verification of the cancer.

Remembering the above conditions, the following case is quoted.

M. B., male, brakeman, age 53 years, married.

Previous Medical History.—Has always been well up to the time of the injury, with the exception of the usual childhood diseases.

Family history is negative with regard to cancer or other malignant tumors.

History of Present Illness.—On June 20, he was walking along a railroad track when a train backed slowly into him knocking him down along the railroad siding. He had many contusions all over his chest and abdomen. He did not stop work after the accident, but did start to complain at once after the accident of having abdominal pain. He lost steadily in weight. After losing much weight and having severe abdominal pain, he went into a hospital where the history record shows that there was a definite mass in the epigastric region and from the X-ray examination a diagnosis of carcinoma was made.

Operation.—September 25; median incision; a large carcinoma was found in-

volving the transverse colon, but apparently not involving the mesentery glands or other parts of the gastro-intestinal tract. Transverse colon was resected. Histological diagnosis was made of carcinoma.

The man recovered and was discharged from the hospital; but did not gain weight, and died January 25.

In comparing this case to the seven so-called guarantees necessary to establish the connection between the injury and the tumor, it is found that this case fulfills nearly all of them.

First, there is no doubt as to the authenticity of the trauma.

Second, the trauma was sufficiently severe to at least slightly injure the abdominal contents.

Third, the integrity of the part prior to the injury cannot be told, as the man had not had a physical examination for many years prior to the injury, but his own statement and the statement of witnesses is that he was perfectly well, had not lost weight, and was doing hard work up until the time of the injury.

Fourth, carcinoma of the transverse colon, while it did not correspond exactly to the site of the injury, did correspond as much as any of the cases recorded, and this statement could not be proved either way before Court.

Fifth, the tumor did appear at a time not too remote from the time of the accident to be reasonably associated with it. Some writers have stated that a carcinoma may appear in anywhere from six weeks to a year and some of them have even given three years.

Sixth, the history shows that there was definite presence of pathological manifestations in pain and loss of weight up to the time of the appearance of the tumor.

Seventh, there was histological verification of the cancer after operation.

SYPHILIS WITH AN EXTRA GENITAL CHANCRE ON THE THUMB, COMING UNDER THE COMPENSATION ACT

J. J. H., age 28, male, single. Has three brothers and two sisters; two brothers married and not living at home; one brother and two sisters are living at home and are all well. His mother is living and well; father died eight years ago as the result of an accident.

Previous Medical History.—Had the usual childhood diseases and has been entirely healthy since.

Present Illness.—September 12, this man scratched his right thumb with a piece of hoop iron. He was the foreman in the shop and gave this cut first aid himself, washed and dried it with a roller towel in the wash room, and then applied

iodine. This cut healed and he paid no more attention to it until about September 26, when he noticed it was swollen and red, and that he had some large glands in the axilla. He then went to the plant doctor who opened the finger. On or about October 17, he noticed that he had a lot of "pimples" over his face, chest and abdomen.

Examination in our Clinic on October 29 showed that on the right thumb on the outer side there is an open sore with indurated edges, covered with a typical, grayish slough, not draining pus. The sore had every appearance of being chronic and had the appearance of a typical chancre. Over his face, chest, abdomen, back and arms he has a diffuse papular eruption which does not itch or hurt.

Teeth are in fair condition. Tonsils not enlarged or red. Throat negative. Heart, lungs and abdomen negative.

Genito-urinary organs on a careful examination show no signs of a venereal disease and show no scars of a previous disease. Inguinal glands are not enlarged. The Wassermann test was four plus with a plain alcohol antigen and four plus with a cholestrinized antigen. Urine was negative except for a large trace of indican.

Diagnosis.—Syphilis with extra genital chancre on the right thumb.

Comments.—As far as the above history goes, it seems fairly definite that this man contracted his syphilis at the time of the original injury. The period of primary incubation, that is, between exposure and appearance of the chancre, is usually about twenty-five days. In his case it was fourteen days, which is not out of the ordinary. The period of secondary incubation, that is, the time between the appearance of the chancre and the advent of secondary symptoms, is usually about six weeks. In his case, it was about five weeks.

In view of the above, the insurance company did not even attempt to fight the man's claim for compensation due to syphilis, and he is now under treatment for syphilis and is drawing compensation, although it is expected that he will return to work after the first month of treatment.

It was noted in his treatment that after he had taken mercury for but a few days the secondary eruption rapidly grew worse and he had a typical Herxheimer reaction. He is now being treated with mercury by mouth and weekly intravenous injections of .9 neosalvarsan.

This case emphasizes only too well the importance of the hygiene of industrial surgical plants, for one case such as this, with a subse-

quent treatment and probable future complications would more than pay the salary of a part-time plant doctor to supervise the hygiene and to make periodical examinations of employees. There seems to be little doubt from the careful study of the source of the infection in this case that has been made, that this man was inoculated from a contaminated common towel in a dirty wash room into an injury that he had received while at work.

There is little excuse for such condition of affairs; yet the fact remains that this man can do nothing but draw about a month's compensation for a serious condition that was not due to any fault or neglect of his own.

BULLETINS TO THE MEDICAL PROFESSION OF THE STATE

By JOHN W. MOWELL, M.D.,

Formerly Chairman Washington State Medical Aid Board.

FRACTURES INVOLVING THE HUMERUS, GLENOID CAVITY, CLAVICLE, ASTRAGALUS AND OS CALCIS, ILLUSTRATING DIFFERENT METHODS OF CARING FOR SAME

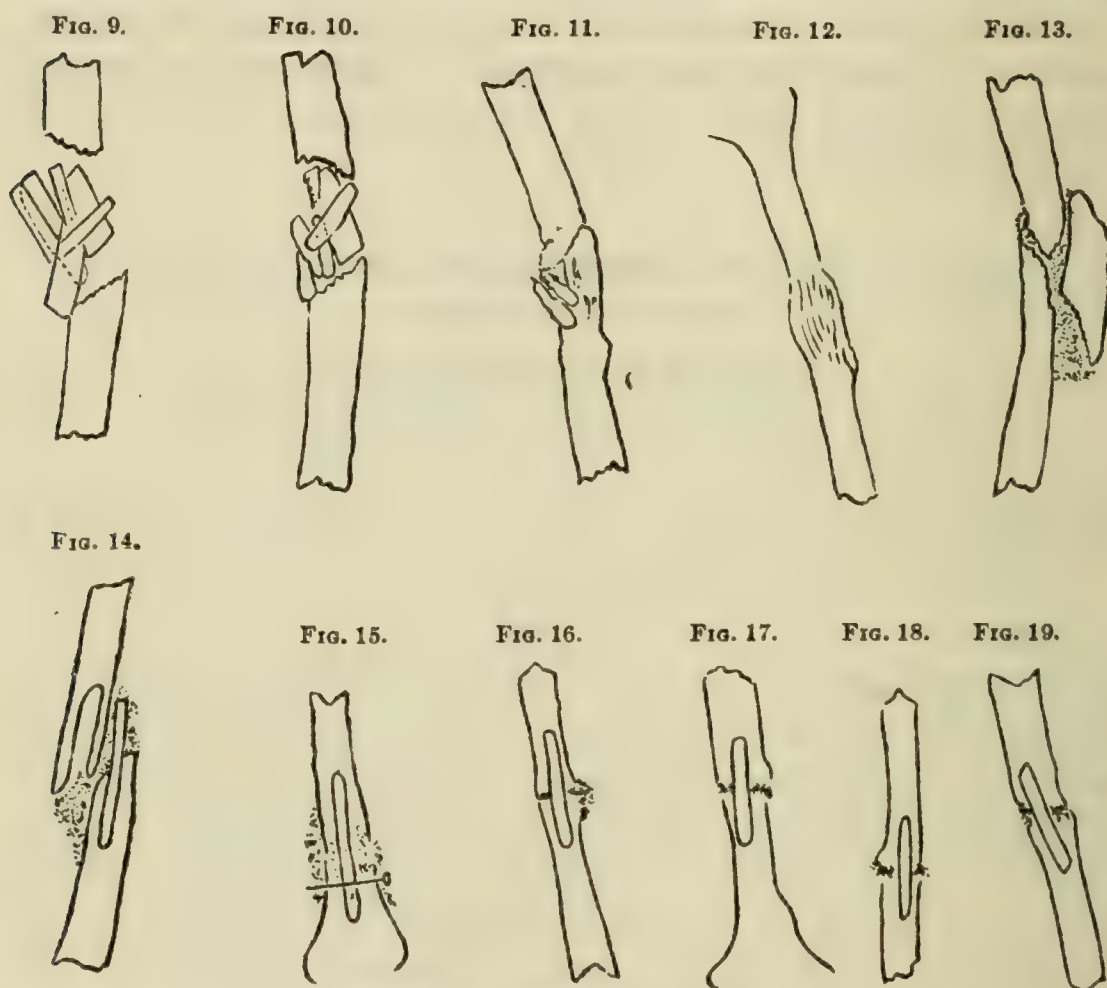
In referring to fractures involving the shaft of the humerus we want to call special attention to the necessity of extension maintained throughout the treatment the same as in fractures of the thigh.

Extension in the treatment of fractures of the shaft of the humerus is just as imperative as in any long bone, and we, herewith introduce Figs. 9, 10, 11 and 12 to illustrate what can be done in compound comminuted fractures of this bone by extension applied that will maintain the arm in normal position and normal length, if applied in the *first* dressing.

Fig. 9 represents a fracture of humerus through the middle third. This fracture was produced through the man's arm being run over by a car wheel—compounded—circulation not destroyed. You will note that there are seven pieces in this fracture broken off at both ends. This arm was immediately put in extension, the skin wound was sewed loosely and maintained at full length as shown three months later in Fig. 10. Fig. 11 shows the same arm nine months later at which time the man went to work. Fig. 12 is the arm fifteen months after

the accident. The line is perfectly straight and practically normal; humerus full length. This man had practically no disability.

The other side of the treatment by *non-extension* is well represented in Fig. 13. Here we have a comminuted fracture that was not compounded, and there was no extension used; note the result. The muscles pulled the ends of the bone together, crowded the piece out, which united, however, as shown here leaving the humerus $2\frac{1}{4}$ inches



Fractures of middle third humerus.

short, with an arm that is seemingly bowed outward and a large amount of bony callus in addition to the piece of the humerus laying on the outer side of the shaft. This interfered greatly with the action of the muscles and this arm was badly disabled. This condition could have been entirely prevented by the same treatment employed in Fig. 9, but it didn't happen to be in the hands of the same surgeon.

Fig. 14 shows a bone inlay that was properly made but undoubtedly slipped out of the groove while the plaster was being applied. However, nature came to the rescue and promptly united this arm. I call

your attention to the well-marked callus along the proximal end of the bone graft. When this radiograph was taken there was well-formed callus and the bone seemed solidly united, the callus extending along both sides of the end of the inlay that was outside of the groove in the shaft. There are statements to the effect that callus does not form along inlays.

FIG. 20.

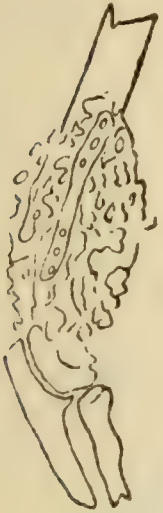


FIG. 21.



FIG. 22.



FIG. 24.



FIG. 23.

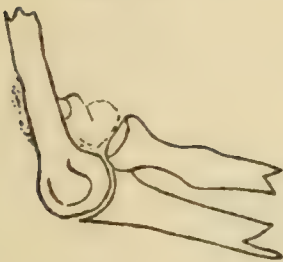


FIG. 25.



Fractures of lower third humerus.

Figs. 16, 17, 18 and 19 show bone inlays. These were all successful operations. You will note the small amount of callus, but solidly united.

Fig. 15 shows an inlay with a nail driven through the distal end, I suppose in order to keep the bone graft from slipping out. We call your attention to the excessive amount of callus, in our opinion caused by the irritation from the nail, because we see this same thing happen

where plates are used *when* there is callus formation. Where non-union is brought about in the presence of a plate, callus formation seems to be entirely wanting, as is plainly shown in Fig. 25.

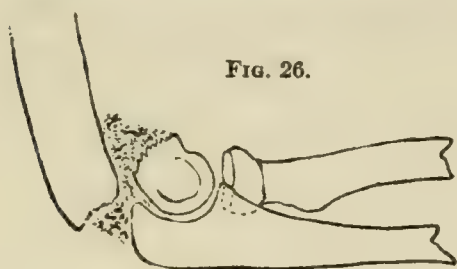


FIG. 26.

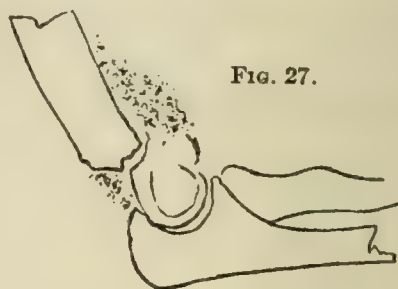


FIG. 27.

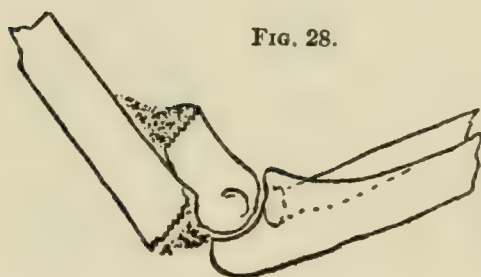


FIG. 28.

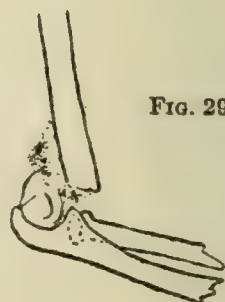


FIG. 29.

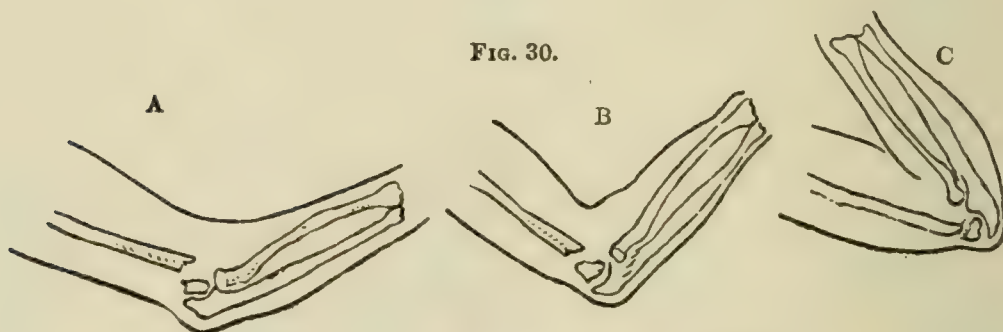


FIG. 30.

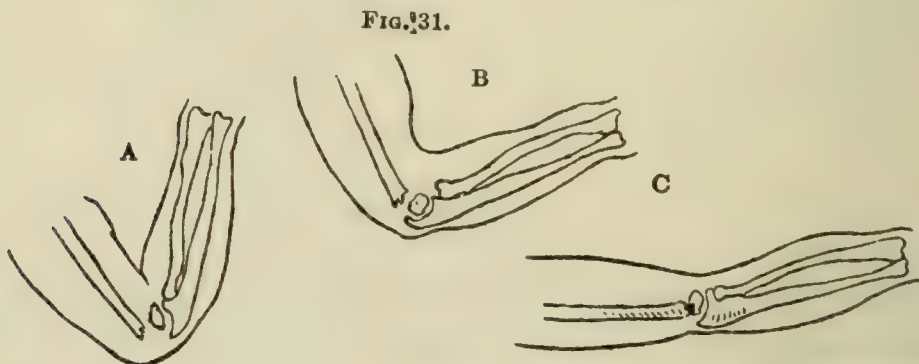


FIG. 31.

Supracondylar fractures of humerus.

Observation under the Industrial Act convinces us that almost any kind of autogenous bone graft, no matter whether it fits or not, is successful. On the other hand, plating, as a rule, is a failure, and eight out of every ten plates that are used in the state are breeders

of trouble only, for the surgeon and more especially the patient, and require two operations as a minimum and often four or five.

We show in Figs. 20, 21 and 22 three arms that were fractured

FIG. 32.

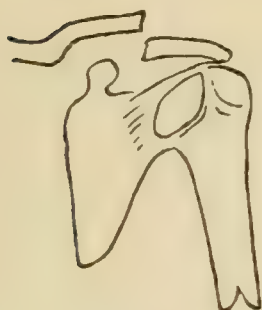


FIG. 33.

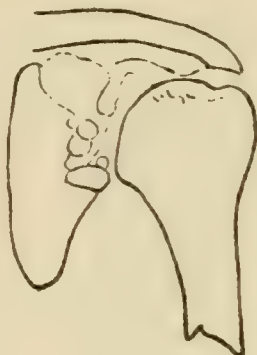


FIG. 34.

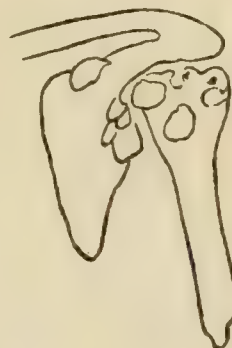
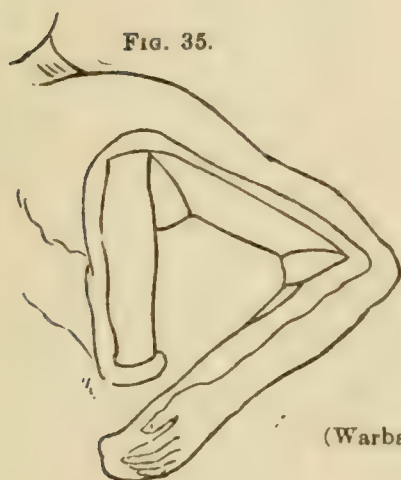


FIG. 35.



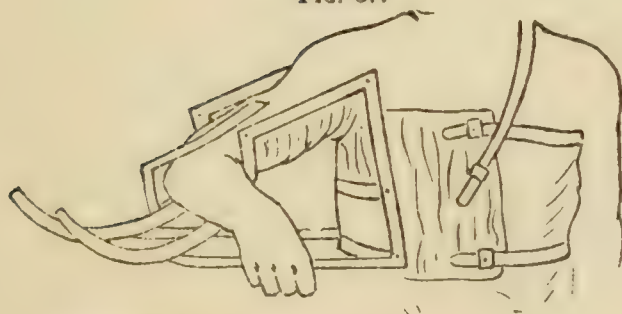
(Warbasse)

FIG. 36.



(U. S. A.)

FIG. 37.



(Warbasse)

FIG. 38.



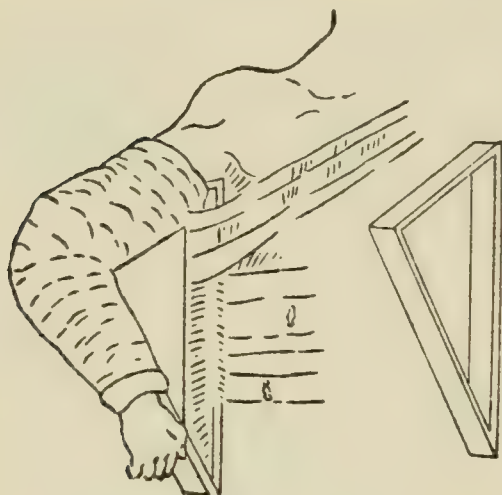
Jones—modified.

Fractures of glenoid cavity and of the head of the humerus.

at the junction of the middle with the lower third, or through the lower third. These three arms were plated. You will note in Fig. 20 plate still in and the ends of the shaft have disappeared entirely

where the screws were placed and replaced by a great mass of tissue of a bony nature. This arm presented a large spindle-shaped swelling of the arm five months after it was plated. Sections of it proved

FIG. 39.



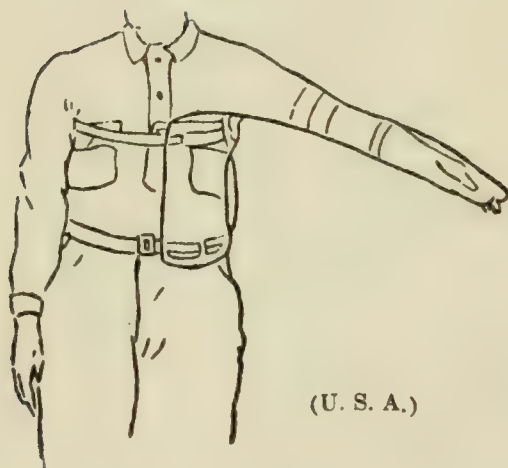
(Hull)

FIG. 40.



(Jones)

FIG. 41.



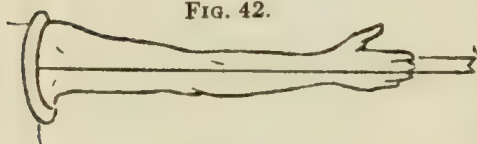
(U. S. A.)

FIG. 43.



(U. S. A.)

FIG. 42.



(Jones)

Abduction splints.

to be osteosarcoma. The arm was amputated at the shoulder, the man returned to Europe and was lost from observation. This is practically the same history as the case presented here in Fig. 21. You will note, however, that this plate is badly bent, screws all loose, and when this man presented himself at the office for examination the

distal end of this plate had cut through the skin by pressure, and a bloody discharge was coming through the opening. This also proved to be osteosarcoma. The arm was amputated and this case was lost sight of as he returned to Europe also. Fig. 22 is practically the same kind of a case, followed the same course. We call your attention to the fact that the distal end of the humerus and condyles have completely disappeared and are replaced by a large bony mass. At the time the radiograph was made the plate had been taken off. This case also proved to be osteosarcoma.

Doctor Murphy, of Chicago, has said many times in his clinics that osteosarcoma never follows fracture. The general opinion is that sarcoma comes from trauma or irritation, so we can come to one conclusion only, that is, these three osteosarcoma cases were a direct result of the irritation from the plate, because we have only had one other case of sarcoma in the arm following trauma, and that sarcoma did not involve the bone, but involved the muscle. It followed in about three months after a comparatively moderate trauma to the soft parts. However, it was near the elbow in the forearm.

Figs. 23 and 24 show a splitting of the shaft of the humerus between the condyles. They were never properly reduced.

Fig. 23 is rather a common injury. You will note that the condyle is carried forward and upward, carrying the head of the radius with it.

Fig. 24 was in a boy about eighteen years of age. You will note that the shaft of the humerus was split and also fractured through the ulna side of the shaft. The radius was carried forward and dislocated. This arm was allowed to unite as shown in this radiograph in full extension. The boy's arm was perfectly straight and completely ankylosed. This left the boy with a useless arm. This was before the Medical Aid Board was created. To-day if a case of this kind would arise we would try arthroplasty to improve this boy's condition, as it is certainly a shame to leave a boy of eighteen with an arm like this.

SUPRACONDYLOID FRACTURES OF HUMERUS

These are very common fractures, of which we have seen a great many. We illustrate four actual cases, because they represent the different types that we see.

FIG. 44.

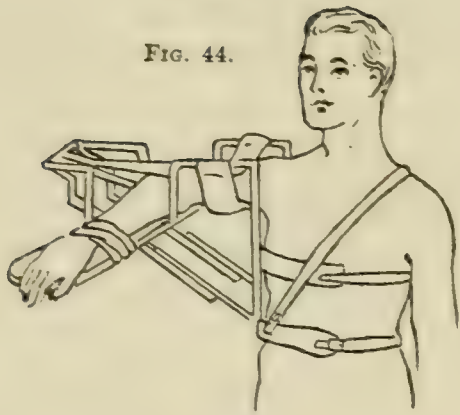
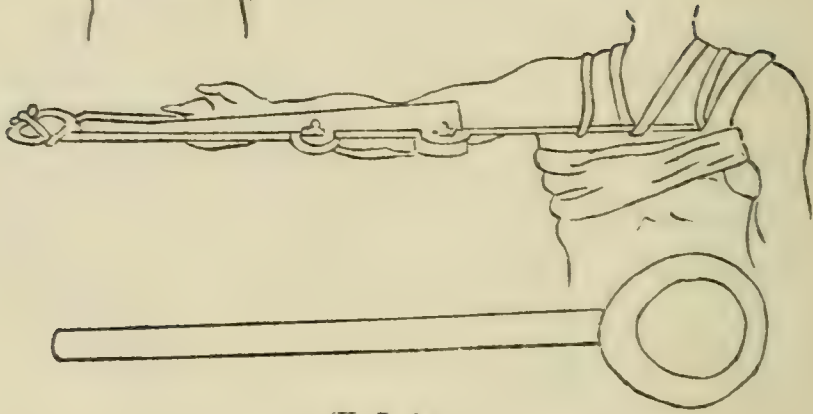


FIG. 45.



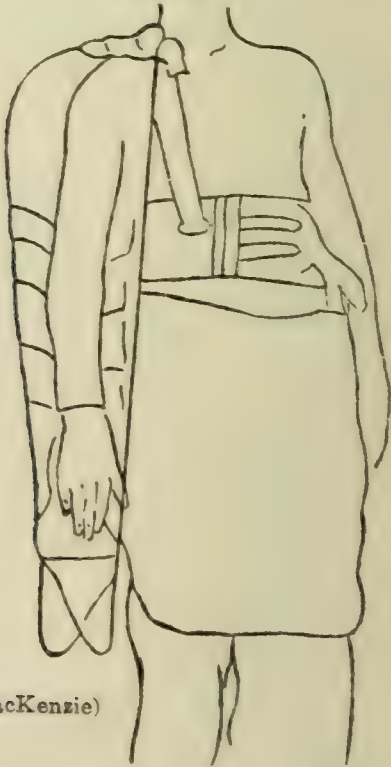
(U. S. A.)

FIG. 46.



(MacKenzie)

FIG. 47.



(MacKenzie)

Fig. 26 was treated by a right angle splint applied to the extensor surface of the arm with no extension. At the time that this man came up for settlement this radiograph showed the present condition. The arm was fixed at a right angle with not more than 10 degrees of movement. You will note the callus poured in between the shaft of the humerus and the olecranon; it was also poured down into the joint, as no passive motion was used to iron it out of the joint while plastic.

Fig. 27 is practically the same condition treated in the same way—same result. If either one of these arms had been put up in complete flexion, or what is known as the Jones position with extension at the time, the condyle would have dropped into normal position, that is, if it had been put up *early*.

We call your attention to Fig. 29, because in this case we have the angle of the fracture run across the humerus in the other direction, which throws the distal end back of the shaft. This arm was treated in complete extension and in the final result the man could extend the arm completely, but he could not flex it any more than is shown in this radiograph, and not to a right angle. This arm should have been dressed in the Jones position, because complete flexion would have pulled this arm into normal position. See illustrations, Figs. 30 and 31, taken from Warbasse's "Surgery" and in Fig. 30 a, b and c, illustrates this same fracture as illustrated in Fig. 29. Fig. 30 a shows what extension does to this fragment and c shows what flexion does.

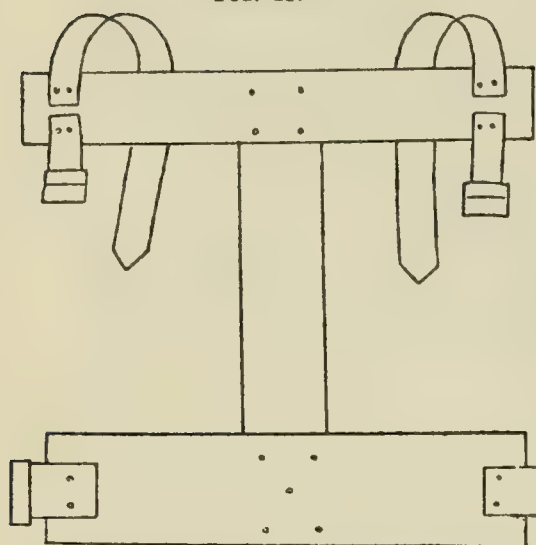
Fig. 28 we have a fracture through the middle of the distal third of the humerus with the distal end overriding on the flexor side of the humerus. In this fracture the fragment is too long to be brought into position by any of the methods shown in 30 and 31 and must be treated by extension applied to the distal fragment by methods shown in Figs. 39, 40 or 43. This man had complete flexion as an end result, but could not extend his arm beyond a right angle, as you will note excessive amount of callus between the distal end of the humerus and the olecranon.

FRACTURED HUMERUS AT MIDDLE THIRD

Fractures of the humerus that occur just below the insertion of the deltoid muscle are fractures which should receive special attention,

because in this fracture we have the upper fragment abducted, and this is the end of "the bone which we cannot control." The distal fragment can be controlled and must be placed in apposition to the frag-

FIG. 48.



All parts padded with felt orthopedic buckles.

FIG. 49.

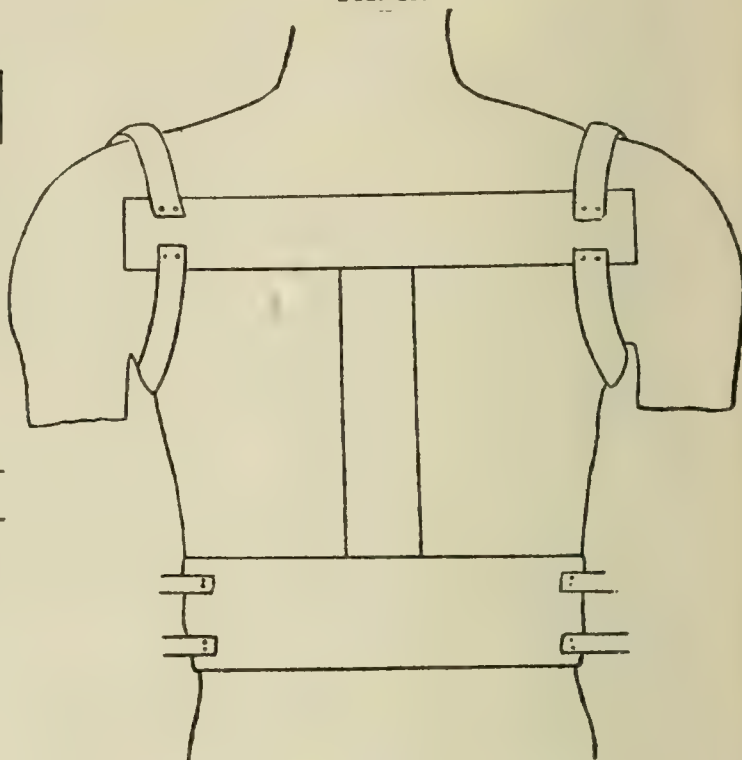
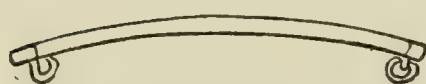


FIG. 50.



Barrel stave splint for fracture and roller bandage.

FIG. 51.



FIG. 52.



Bodine modified by Bird.

FIGS. 48 and 49.—Splint for treating fractures of the clavicle.

ment that we cannot control. So it becomes necessary to abduct the arm to an angle of about 60 degrees from the body.

In fractures that occur above the insertion of the deltoid the proximal end is usually rotated externally by the supra and infraspinatus muscles, so it becomes necessary to place the arm in external rotation so when union takes place the man has not lost external rotation.

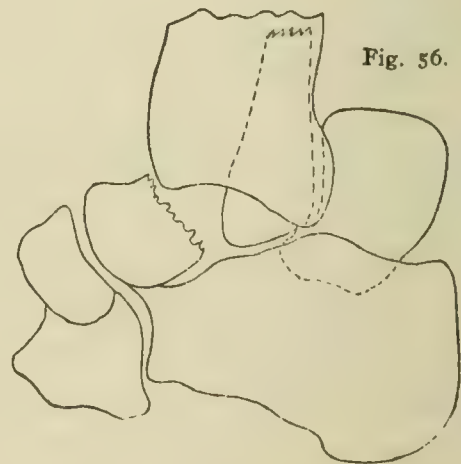
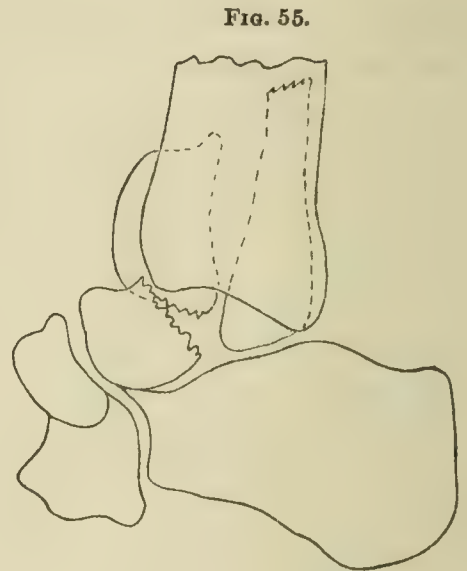
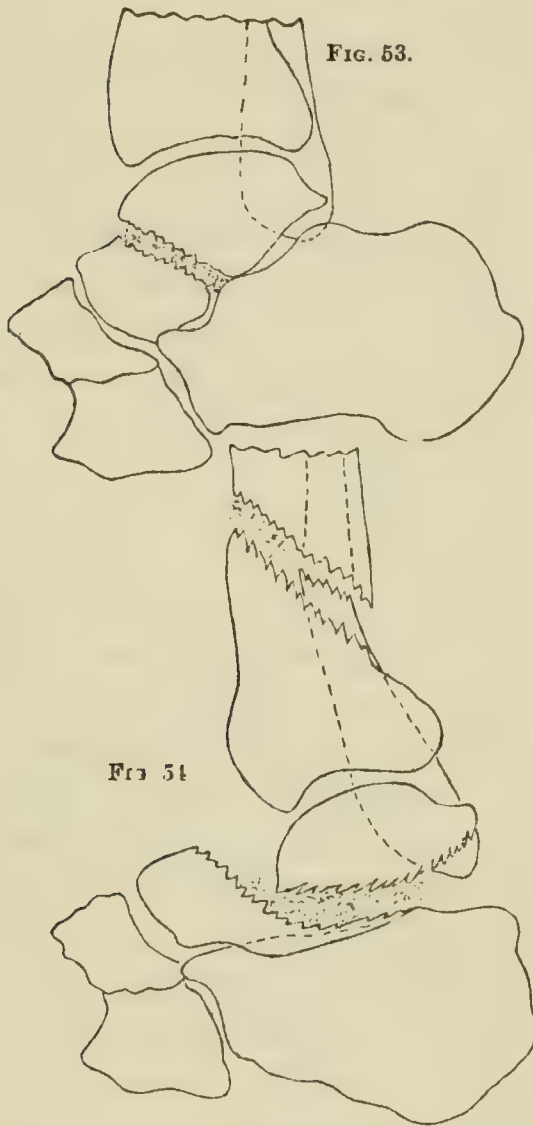
This is best maintained by the splint known as the "airplane splint," which is well adapted to maintaining this external rotation.

In shoulder injuries, especially where the deltoid is involved and the circumflex nerve also, it is best to put the arm up in abduction. This takes the pressure off the bursa in the shoulder joint, which is an important matter. Then it prevents overstretching of the deltoid muscle; it also puts the adductor muscles of the arm in an extended position. This is an advantage in recovery, because the muscle that has the most work to do is kept in the best condition, that is, the one that raises the arm, and it regains its normal strength and function in a shorter period of time. We too often hear the following: "They just put my arm up so (illustrating by placing his arm down by his side flexed at the elbow) and left me alone—doing nothing more—took the dressing off, and said: 'The bone is now united—use it.'" In these cases the surgeon in charge should give the passive motion himself and give directions to the nurse for massage and heat.

FRACTURES INVOLVING THE GLENOID CAVITY AND THE HEAD OF THE HUMERUS

These are fractures that need special care. Figs. 32, 33 and 34 illustrate three cases. Fig. 32 had damage to the glenoid cavity and the head of the humerus—fractured at the surgical neck and split in two pieces. Fig. 33, the man fell, striking the shoulder on pavement, which damaged the head of the humerus and chipped six pieces off the glenoid cavity. Fig. 34 is practically the same injury only it was produced by being struck on the shoulder by a swinging timber. All three of these cases were treated in Sayre's dressing. As an end result we had complete ankylosis at the shoulder joint, that is, between the head of the humerus and the scapula. These three men had abduction of about 35 degrees only. It is a serious mistake to put up any fracture of this kind in a Sayre's dressing or down by the side of the body. Any fracture that involves the glenoid cavity and the head of the humerus should be maintained in an abducted position of about 60 degrees as shown in Figs. 35, 36, 37, 39, 41, 42 or 44. The reason for this is self-evident if you will stop just one moment to think. This is well illustrated in Fig. 38 which is modified from a Jones illustration. The arm as shown here is one that was maintained at 60 degrees abduction during treatment. So in the final

result he had abduction of 120 degrees or almost normal function of the arm, because he has 60 degrees adduction that he would not have had if the arm had been treated in a Sayre's dressing, because adduction and abduction that he gets is brought about by the *excursion* of the *scapula*. An arm maintained at 60 degrees and becoming



Fractured astragalus with dislocation. Fig. 55 shows fracture of the astragalus through the neck with the posterior body of the astragalus dislocated forward. Fig. 56, same fracture with the posterior body of the astragalus dislocated backward, which is the most common displacement.

ankylosed can be placed down by the side, because the scapula will swing in as shown by the dotted line, and on elevating the arm it will swing out as shown by the black line. If this injury is treated by a Sayre's dressing or down by the side the scapula will remain as shown on the right arm of Fig. 38, and it will never assume the posi-

tion as shown in the dotted lines on the left side, and its farthest excursion will be as shown by the black line on the left side, from a position one-half way between the dotted and black line, and will only

FIG. 57.



FIG. 58.



FIG. 59.



FIG. 60.

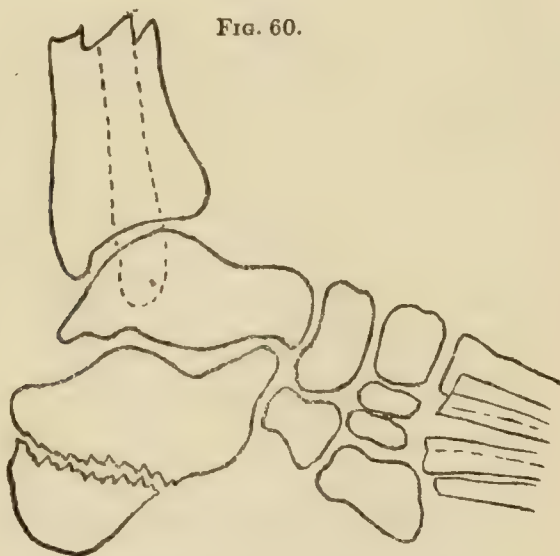


Fig. 57 illustrates one where the man lit on his heel, fracturing the os calcis through its body. A fracture running vertical to the body of the bone comprises about 30 per cent. of the fractures occurring in this bone. About 30 per cent. run horizontal to the body of the bone.

Fig. 58 shows the os calcis fractured at the insertion of the gastrocnemius and is always produced by a man lighting on his toes. In one case the man fell about thirty feet, lighting on his toes, producing this same fracture in each foot.

Fig. 59 shows a combined fracture through the body and also a portion pulled loose by the tendon achilles—all now firmly united, but deformed.

Fig. 60 shows fracture of os calcis and downward dislocation of cuboid. (This case started a law suit.)

allow an abduction of 45 or 50 degrees at the extreme. So we cannot be too emphatic in calling attention to the treatment of injuries of this kind.

SPLINTS

The plaster Paris splint shown in Fig. 35, taken from Warbasse's "Surgery," is one that any surgeon can build on the patient and fits perfectly after adapted to the arm and body—built out of a number of layers of plaster gauze used as a pad.

Fig. 36 is an abduction splint used by U. S. Army for the treating of the same kind of injuries and made of rod wire and fastened to the body of the patient as shown with binders.

Fig. 37 is a standard splint for extension of the humerus and abduction.

Fig. 39 illustrates a very good dressing for a fracture involving the shaft of the humerus and is easily applied.

Fig. 40 illustrates the Jones extension splint for fractures involving the shaft of the humerus. This is a very good form of extension for the humerus. Owing to the pressure made by the ring under the arm it is not comfortable to the patient, but is a producer of good results. It cannot be used, however, for abduction of the arm, and requires a right and left splint as it is not interchangeable. I call your special attention to Fig. 44 known as the "airplane splint"; it has all the advantages of the Jones and has none of its disadvantages. You will note that it has nothing under the arm, but the bars run up over the chest in front of the shoulder and over the scapula in the back, and when applied does not interfere with the circulation under the arm, nor make pressure on the brachial plexus. It also has all the advantage of extension for arms and forearms and external rotation.

FRACTURES INVOLVING THE GLENOID CAVITY AND INJURY TO THE HEAD
OF THE HUMERUS ALWAYS RESULTING IN ANKYLOSIS OF SHOULDER

We call your special attention to Fig. 38 which shows the possible excursion of the scapula by the dark and dotted line. The position shown in the dotted line is not possible, however, when the arm is treated down by the side, but is possible only when the arm is maintained at an angle of 60 degrees. The securing of this additional 60 degrees in abduction is a very important matter, allowing the arm an excursion of 120 degrees instead of a limit of 45 to 50 if treated by a Sayre's dressing.

METHODS OF FIXATION AND EXTENSION FOR FRACTURES INVOLVING THE HUMERUS

AIRPLANE SPLINT AND OTHER EXTENSION APPARATUS FOR FRACTURES OF THE ARM AND FOREARM

Fig. 45 shows the Thomas splint applied to the arm for extension in injury to the humerus. You will note that it is applied over the opposite arm extending across the chest and back and maintained in position by a wide binder around the body and through the ring under the arm opposite to the one to which extension is applied. This entirely obviates the disadvantage brought about by pressure under the arm as shown in Figs. 40 and 42 and is claimed to be a very comfortable dressing for extension.

Fig. 47 illustrates the MacKenzie method of extension. You will note that there is no pressure in the axilla in this extension apparatus and it is an ambulatory splint. It is illustrated here with other methods available, of which every industrial surgeon should be thoroughly familiar, as he must be master of many methods to get results. However, the "airplane splint" as illustrated here is made adjustable and can be adapted to almost any fracture of the arm or forearm.

FRACTURED CLAVICLE

Ever since surgeons were called upon to put up fractures of the clavicle they have followed one principle, that is, of *trying* to maintain the shoulder in an upward and backward position, thereby making extension on the clavicle. Sayre's modified dressing, which is universally followed in the treatment of this fracture, is a poor one for several reasons. First, it is very uncomfortable. Second, it is almost impossible to maintain it as applied. Third, if bandage material is used it invariably slips off and allows the shoulders to assume a downward and forward position. If adhesive plaster is used it irritates the skin so the patient complains bitterly of restraint and is apt to take the dressing off. In my opinion this manner of dressing of fractured clavicles should be abandoned as not being a practical dressing, and has nothing in its favor for the treatment of a fractured clavicle.

All that is necessary to maintain the clavicle in position is a backward pull on the shoulders. The splint shown here in Figs. 48 and 49 is made of light wood with straps attached, the entire splint,

straps and all, well padded, placed on the back of the patient and maintained around the body by the heavy strap at the bottom and the upper end of this "T" placed across the back of the scapulæ pulls both shoulders backward and makes sufficient pull to hold the clavicle in line and maintain it in position through the entire course of treatment. This splint allows the patient the use of both arms and all he has to do is to submit to the restraint placed on him by this splint which pulls both shoulders well back and usually no other dressing is required.

This splint should become a universal dressing for all fractured clavicles, and should be in the hands of every hospital and every industrial surgeon in the state. Even in dislocation of the acromial end of the clavicle this splint and the strap over the shoulder will maintain the end of the clavicle in normal position, and in almost every case will obviate the necessity of surgical interference if worn sufficient length of time. It absolutely prevents any overriding of the clavicle when broken through the middle or distal third.

Fig. 50, barrel stave with roller bandage nailed on the ends and placed across the chest and figure of eight bandage across the back.

FRACTURED ASTRAGALUS WITHOUT AND WITH DISLOCATION OF FRAGMENT

Fractured astragalus without dislocation is a very common injury and is a very important one for the reason that recognition and proper treatment give the man a useful foot often with little disability; but if unrecognized and not treated according to approved methods for this fracture it is very disabling. Fig. 52 shows a simple fracture through the neck of the astragalus. Fig. 54 shows a fracture through the neck and also through the body of the astragalus, and the articular position dislocated backward complicated by a fracture of the tibia and fibula.

FRACTURE OF THE ASTRAGALUS

Fracture of the astragalus is a very common injury and is almost invariably produced by a fall alighting on the feet, especially often where they light on one foot. The milder forms of this injury may result in simple impaction between the articular surface of the astragalus and the os calcis. The most common form is a fracture through

the neck of the astragalus with no displacement. Where the force is more violent, combined with an indirect force, we are very apt to have dislocation of the posterior fragment.

Fig. 56 shows fracture through the neck of the astragalus with posterior dislocation. This fragment may be in the interspace between the tibia and fibula on the posterior surface and just in front of the tendon achilles. This fracture is very commonly overlooked even in the face of good radiographs. Just recently one surgeon in the state had two cases of this kind that were identical. The first man fell some distance striking on his foot. The second man started to run to get away from a flying line; the cable struck him across the plantar surface of the foot. Splendid radiographs were made with diagnosis of Pott's fracture in each case, and treated as such. It has been the experience of the Medical Aid Board that the only thing to do with this fragment, when seen late, is to remove it, which we have done in a numbr of instances with much benefit and improvement in the function of the ankle and the relief of pain. This is the particular fracture that Dean Lewis, of Chicago, states, "if recognized at the time of injury it is impossible to reduce same except in one way only, and that is to do a tenotomy on the tendon achilles." This allows the foot to drop down and the bone falls into normal place, he says, "very easily."

One fracture of this kind within the last year fell in the hands of one of our good surgeons in the state. He attempted to reduce it and failed until he did a tenotomy, and he said it almost reduced itself.

Fig. 55 shows the same fracture except the posterior body is dislocated forward. We have only had two of this kind, in the state, in ten years. You can easily feel the fragment laying on the dorsum of the foot just in front of the fibula and tibia, more properly speaking in the interspace between the two and above the tarsal scaphoid.

One of these men was advised to have this removed but his surgeon told him if he had it removed he would be crippled all the rest of his life, so he declined to have it removed and he is certainly still crippled. In removal of these it is better to take out the entire astragalus so the articular surface of the tibia will come a little farther forward on the foot than it is possible if you leave the anterior fragment in, because the tibia will settle down on the os calcis, and it is too far back so the man does not have good dorsal flexion like he does have when the anterior fragment is removed. After a period of time they have

a pseudo-joint between the os calcis and tibia, also the weight-bearing line is better. In these cases the foot should be well dorso-flexed.

FRACTURE OF OS CALCIS AND DISLOCATED CUBOID

Fractures involving the os calcis are very common and usually result from falls. If a man falls and lights on his heels there are one of two things pretty sure to happen. He either fractures the astragalus, or the os calcis, or both. If he strikes squarely on his heels the os calcis is apt to be fractured through the body. There are a great many varieties and we do not attempt to show any. However, if the man lights on his toes and his os calcis does not receive a direct blow the muscular pull from the gastrocnemius muscle is almost sure to result in a fracture of the os calcis by jerking off the posterior portion where the tendon is attached, of which we show two.

Surgery

DIAGNOSIS—FRACTURE OF THE SKULL

By R. J. BEHAN, M.D.

Surgeon to St. Joseph's Hospital, Pittsburgh, Pa.

IN fracture of the skull, the classical symptoms of fracture, such as crepitation, deformity due to overriding or sepression of the fragments, tumefaction, pain and loss of functions, may be entirely absent. These symptoms, even when present are only of serious concern, so far as they are indications of a possible injury to the intracranial structures with increase of intracranial pressure.

Of the above symptoms, crepitation (unless the fracture is very extensive) is practically never elicited. Deformity in a depressed fracture may be marked immediately after an injury. It is usually quickly disguised by the associated hemorrhage. Careful examination and notes should be made, in all cases of head injury, of the contour of the head at the first examination. For an accurate examination, it is necessary that the head be shaved. Localized tumefaction and swelling is of some value in formulating a diagnosis, and will be considered under the symptoms produced by hemorrhage. At times, as the result of a bad injury, a depression of the bone may occur without fracture. This is well illustrated by an example given by Lockett, who, in a child after a fall in which it struck its head against the floor found a three-cornered depression without fracture, but with symptoms of irritation. Such a characteristic deformity, he thinks, is typical of injuries of the skull, in which the head strikes against a flat surface.

In fracture of the skull (as in fracture elsewhere) pain usually is experienced while the patient is conscious. If the patient is unconscious, he becomes restless and is partly aroused as soon as pressure is made over the fracture. The line of fracture may be defined by making pressure with the finger in various directions. At the point where the patient complains of the most pain, dots are made. These dots are then connected by a line. This line corresponds fairly well with the line of the fracture.

Auscultatory percussion, as devised by Murphy, may also be used to confirm the direction and location of the line of the fracture.

The principle underlying this method of auscultatory bone percussion being, that through intact bone, the percussion note is transmitted more clearly, to the bowl of a stethoscope, placed at a distance from the area percussed than when a dissolution of continuity in the bone is present. A very small bowl stethoscope should be used, and the percussion should radiate toward it; as soon as the fracture line is passed the note becomes much clearer.

Loss of or disturbance of intracranial originating functional activities may be a direct or an indirect resultant of fracture of the skull, and is the most serious of the pathologic sequences of such a fracture.

For a working basis, it is best to divide the symptoms of fracture of the skull into three groups.

I. Those due directly to the traumatism. These are immediate and remote.

II. Those due to brain compression, either from pressure by the bone fragments or from pressure by hemorrhage external to the brain cortex.

III. Those due to injury of skull contents; brain membranes as dura or pia-arachnoid, brain cortex and subcortical structures.

I. TRAUMATISM

Symptoms due directly to the traumatism referable to the head are immediate and remote. The immediate resultants of head traumatism are—(a) *Loss of consciousness*. Immediate loss of consciousness results from concussion. This unconsciousness state may be momentary or may be prolonged, depending upon the force of the blow, the direction of the blow and the point of impact. A blow on the back of the head seems to produce more severe reaction than does one on the front of the head. In some cases the blow may not be sufficient to cause a loss of consciousness, but may be severe enough to momentarily stun the recipient, so that he becomes "groggy." He may lose his equilibrium and may fall. He may also become nauseated and may vomit. The sympathetic nervous system may also be disturbed and the patient's skin is blanched. This state may persist for only a few minutes and merge into the state of brain compression or it may be quickly relieved, after which the patient usually complains of a severe headache localized to the area traumatized.

(b) *Bruising and contusion of the scalp.* A blow upon the head will frequently lead to bleeding into the pericranial tissues with ecchymosis. The bleeding frequently occurs in the subperiosteal cleft. Swelling due to bleeding from the diploe of the bone into this space is limited to the suture lines. This limitation is of important diagnostic significance because, a swelling definitely delimited by the suture lines is pathognomonic of a fracture of the skull.

(c) *Laceration of the tissues of the scalp.* If the blow had been severe enough and at the proper angle, a clean cut, sharp margined wound, similar to an incised wound may result, and may extend down to the pericranium which as a rule is not lacerated. In other cases irregular destruction of the superficial tissues may result.

The remote resultants of head traumatism are—functional brain disturbances, *viz.*—disturbances of mental perception and character, irritability, emotionalism, instability, headache with epilepsy as an heritage.

II. COMPRESSION OF THE BRAIN

(a) Compression of the brain may be due to direct pressure by depressed bone fragments of the fractured skull. If the fracture is very extensive and the blow was of great severity, the bone may be comminuted, and the fragments be driven deep into the brain substance or be deeply depressed, so that considerable compression of the brain results.

(b) Compression of the brain may also result from hemorrhage (oozing from the diploe of the bone or from ruptured meningeal or pia-vessels). The sinuses and larger veins are also occasionally ruptured. Bleeding from the diploe of the bone may be in two directions.

1. External to the skull, underneath the pericranium. This type of hemorrhage is not pernicious and causes no serious disturbance.

2. Internal between the skull and the dura, or between the dura and the brain cortex.

The hemorrhage resulting from fracture of the skull may be arterial or venous in origin. Arterial hemorrhage is the result of a tear or rupture of the middle meningeal artery or of one of its two branches; or may result from injury to the arteries in the pia-arachnoid. Middle meningeal hemorrhage is a very serious event

and may lead to death, unless it is at once controlled. Should the hemorrhage be subdural, it is extremely dangerous and death may, rather quickly, occur. Arterial bleeding, however, is more likely to be extradural. Both types of hemorrhage cause definite symptoms which will be later fully discussed. If the bleeding continues, until the intracranial pressure is raised beyond the point of tolerance, *i. e.*, beyond the point where the circulatory system can maintain a sufficient blood-pressure to overcome the increasing intracranial pressure, resulting from the blood clot, so that the circulation to the medulla is unimpaired, death occurs. Arterial hemorrhage may also come from the arteries of the pia-arachnoid, even without a tear of the dura. Pia-arachnoid arterial hemorrhage is apt to prove rapidly fatal, because the cerebrospinal fluid retards blood coagulation and therefore the diffusion and spread of a pia-arachnoid hemorrhage is unhindered.

Venous hemorrhage, if it occurs in the closed pia-arachnoid space, is especially dangerous, but occurring in the epidural space usually does not lead to a fatal issue, because the pressure from an intracranial clot of venous blood cannot raise the intracranial pressure to a dangerous degree. Hemorrhage from the sinuses is very serious. Infiltration of the cortex may result and death is the usual termination.

Intracranial traumatic hemorrhage may be classified according to location as

- | | | |
|---------------|-----------------------------------------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| (a) Meningeal | { Extradural
from the

Subdural
from the | { a. Middle meningeal.
b. Dural sinuses.
c. Diploic vessels.

a. Meningeal arteries.
b. Sinuses.
c. Pial vessels.
d. Hemorrhagic pachymeningitis.
e. From extradural sources, when the
dura is torn.
f. Laceration of brain terminals of
middle cerebral from rupture of
cerebral or cerebellar vessels. |
| (b) Cortical | { Thalmic.
Subthalmic.
Ventricular.
Capsular.
Pontine.
Cerebellar. | |

Excusing the repetition, because of emphasis, I shall repeat that the bleeding, from a fractured skull, may be either external or internal to the skull.

Bleeding externally between the skull and the pericranium gives rise to swelling. The swelling of the skin is most marked over the line of separation of the fragments, and gradually decreases toward the sound tissue.

The limitation of the swelling is explained by the above drawing. It is seen that blood comes from the fractured bone at A and gradually extends in the pericranial space, toward the periphery C and B. It is to be noted that the direction and extent of the blood clot gives some indication of the site and the direction of the fracture. This type of bleeding is slow and the swelling develops gradually and causes no ecchymosis. It also has not the well-marked ring of abrupt induration as is found in subfascial hemorrhage, and is limited by the suture lines.

If the pericranium is ruptured, hemorrhage may occur in the subaponeurotic space. The extension of this hemorrhage is limited by the line of attachment of the tendinous aponeurosis of the muscles to the bone. Hemorrhage beneath the temporal muscle extends to the superior line of the occipital bone, posteriorly, while anteriorly, it extends to the orbital arches, and inferiorly as far as the zygomatic arches. Large hemorrhages are, as a rule, rare.

If the muscular aponeurosis is ruptured, hemorrhage may appear under the skin, or, if the skin is also ruptured, it may appear on the surface as a free discharge of blood or may infiltrate through normal barriers into the periorbital fat.

Free hemorrhage may also occur in regions of the skull where there is no restraining pericranial or fascial barrier, such as in the orbit, the nose, the middle ear. Hemorrhage into the orbital fat occurred in 6 out of 95 cases in which there was no fracture of the roof (Holden quoted by Maggruder). Hemorrhage into the nose may occur through fracture of the horizontal plate of the ethmoid, or through the eustachian tube into the nose, when the hemorrhage is from a fracture of the middle ear. When fracture of the petrous portion of the temporal bone is present, the sphenoid may be fractured and give rise to pharyngeal hemorrhage. Throat hemorrhage may result from fracture of the base of the skull.

The location of the fracture of the skull may be indicated in many instances by the occurrence of ecchymosis at a definite locality as,

1. Ecchymosis over the mastoid area occurs in fractures of the middle and posterior fossa of the skull.

2. Subconjunctival ecchymosis results from hemorrhage within the dura of the anterior fossa of the skull. The hemorrhage spreads along the optic nerve into the orbit. Sometimes, the blood accumulates in orbital fossa and pushes forward the globe of the eye, so that an exophthalmos results.

3. Ecchymosis occurs in the neck and at the base of the skull, when fracture of the occipital bone is present.

III. BLEEDING INTERNAL TO THE SKULL

1. *Between the dura and the skull.* When bleeding between the dura and the skull takes place, the blood gradually spreads out over the surface of the dura and the clot slowly increases in size. Its extent finally is limited by the dural reflections, such as the falx cerebri, falx cerebelli and tentorium cerebelli. Frequently before this occurs, however, the increased intracranial pressure has caused the death of the patient. The symptoms which are present in such conditions are the result of the increase of intracranial pressure. The symptoms, at first, are those of cortical irritation and later of brain compression. The stage of cortical irritation is ushered in by delirium and excitability, gradually decreasing powers of perception (from dullness, apathy and stupor, to unconsciousness and coma which finally deepens into death). Coma occurs when intracranial pressure is sufficiently high to produce a circulatory stasis; in other words, it must be as high as the systolic pressure in the arteries supplying the brain. According to Pagenstecher (Oppenheim, p. 749) coma occurs when the hemorrhage exceeds 37-40 cm. hg.

The period between the injury and the onset of disquieting symptoms such as coma, paralysis, ocular disturbances such as contraction or dilation of one pupil or deviation in the movement of the eye-ball is called the *free interval* or *free period*. The free interval is the time required for the hemorrhage to separate the dura from the inner surface of the skull, or the dura from the brain cortex. In infants, the dura is closely applied and intimately adherent to the inner surface of the skull; consequently, extradural clots are rare.

The slow progress of extradural clot may also be due to the fact that shortly after the injury, the patient suffers from concussion of the brain and from shock, with consequent low blood-pressure. Because of the low blood-pressure, there is less tendency to bleed.

The free interval also may represent the period required for the seepage of blood through the fracture line into the epidural space. After a clot has once formed, it may be increased by secondary hemorrhage which may come on at varying intervals. These give rise to a repetition of or an increase in severity of symptoms already present.

2. Hemorrhage between the dura and the brain cortex, *i.e.*, from the pia-arachnoid vessels either with or without epidural hemorrhage gives rise to:

(a) The symptoms as above described, except that the lucid interval is very short. The irritative symptoms, such as restlessness, twitching, convulsive movements, etc., are very severe and more generalized than in the epidural hemorrhages. The symptoms, however, develop more slowly. (Oppenheim, p. 750.) A positive and absolute diagnostic sign of subdural hemorrhage is the presence of blood in the cerebrospinal fluid. This is revealed by spinal puncture.

(b) Blood is absent in the cerebrospinal fluid in epidural hemorrhages and is the great differential test between the epidural and subdural hemorrhage. The cerebrospinal fluid may be of a brownish yellow tint, due to admixture with some elements of blood. The presence of blood may be confirmed by the microscope or by chemical reactions. The normal pressure of the cerebrospinal fluid is, according to Krönig, 125 mm. of water with the patient on the side, 410 mm. on sitting. It normally is clear and contains 0.2 to 0.5 per 1000 per cent. albumin and about 3 or 4 cells in a field 400 times magnification.

IV. SYMPTOMS DUE TO INJURY OF THE CONTENTS (BRAIN) OF THE SKULL

The symptoms which are the result of injury of the brain vessels vary, depending upon the localization of the lesion in the brain. The regions of the brain, injury of which produce definite symptoms complexes are (A) cerebrum, (B) cerebellum, (C) pons, (D) medulla. Ventricular affections also give rise to a definite symptom complex,

but as ventricular hemorrhage is most usually associated with apoplexy, I shall not consider its symptomatology.

A. The *cerebral symptoms* may be divided into sensory and motor.

1. The sensory symptoms are rather indefinite. They vary from a peculiar hazy change in touch and impressions associated frequently with formications to major disturbances, where pain is the response to touch impressions. In severe cases, the sensory disturbances may go beyond this threshold and all sensation may be abolished over the certain area; even over the entire half of the body. This is termed hemianæsthesia.

2. The motor symptoms are due to central disturbance of the upper motor neuron. When the upper neuron (cortical cells, Rolandic area) is hypersensitive, there results increased superficial reflexes and possibly convulsive movements. Finally, if the irritating persists and increases, paralysis with *increase* of the deep reflexes (Babinski toe reflex) and loss of superficial reflexes ensues. It is characteristic that the motor symptoms are first localized and then become general. Aphasia is present when the lesion is on the left side. The paralysis of the respiratory muscles on the side opposite the lesion is a valuable sign, especially in cases of coma.

The motor localizing signs do not develop immediately, but, as a rule, at some point in the following complex of symptoms:

- (a) Pain in the head.
- (b) Restlessness.
- (c) Twitchings (muscular).
- (d) Dullness of intellect.
- (e) Somnolence.
- (f) Slowing of pulse.
- (g) Stupor.
- (h) Slow, deep respiration.

When motor symptoms do occur, they are of value in localizing the lesions to a particular area of the brain. The chief motor localizing complexes of symptoms are:

- (a) Spasms of a group of muscles.
- (b) Increased reflexes of opposite side with hypertonus.
- (c) Convulsions. The earlier the localizing symptoms de-

velop in the course of the above, the nearer is the hemorrhage to the localizing area.

In this monograph the location of the different centers in the cerebrum will not be considered. Their location is described in any physiology.

In fracture of the skull or in traumatisms of the head, not severe enough to cause fracture, damage to the brain may occur at a point distant to the place where the impact of force was received. It is rather confusing to find localizing signs pointing to a part of the brain than the area of the brain under that part of the skull, where the impact of the blow was received, or as suffering injury. For instance, the blow may have been received on the left side, but the symptoms point to a cerebral injury on the opposite. At the same time, there may be a paralysis of the facial muscles on the right, indicating an injury of the facial nerve. In this case the brain injury was on the left side and the fracture passed through the petrous portions of the temporal bone.

When the blow is on the vortex of the skull, the force of the blow may be diffused at the base of the frontal lobes.

Fracture into the longitudinal sinus is characterized by immediate *spastic paralysis* of the legs, frequently associated with spastic paresis of the approximal segments of the upper limbs. The cause is, according to Makins, occlusion of the superior, longitudinal sinus or of the veins that enter it by fragments of a depressed fracture of the vertex.

B. Cerebellum.—Injury causes an increased pressure which produces vertigo, nausea, vomiting. Cerebellar disturbances also give rise to cerebellar syndromes as

1. Disturbances in reflex and tonic control. (Pendular).
Ipsilateral areflexia or hyporeflexia. (K. J.) Ipsilateral atonia or hypotonia.
2. Disturbances in volitional control, ipsilateral asthenia.
(Affected side is moved less easily and extensively.)
3. Disturbances in coördinative control.
 - (a) *Adiadochochinesis*.
 - (b) *Dysmetria* (Rebound).
 - (c) Migration and staggering toward side of lesion.
4. Disturbances in ocular movements.

- (a) Nystagmus of fixation. (Slow movements to side of lesion lasts for months.) May be diagnosed from labyrinthian nystagmus which lasts a short time, not more than a week. The nystagmus is nearly always horizontal. The slow component toward side of the diseased labyrinth.
- (b) Skew deviation.
- (c) Conjugate deviation.
- 5. Disturbances in spatial orientation.
- 6. Disturbances in sensations (none are present). If sensory disturbances are found, a pure cerebellar lesion is not present. (Multiple sclerosis may be present.)
- 7. Disturbances of speech—scanning, explosive, lisping.
- C. *Medullary*.—Medullary injury as a rule is very serious, as it affects the vital centers.
 - 1. Cardiac centers. Stimulation of the vagus, at first slow pulse, then as the pressure increases paralysis of the vagus causes a rapid pulse.
 - 2. Vasomotor centers. Stimulation of these centers is the result of reflex action or is the resultant of an effort to keep up the circulation in the medulla. Pressure on the medulla and other parts of the brain, beyond a certain degree, gives rise to paralysis.
 - 3. Respiratory centers, when first stimulated (through vagus) cause deep breathing and then slow breathing. On further stimulation, the associated respiratory centers cause more rapid breathing. Then, further stimulation associated with paralysis produces Cheyne-Stokes breathing, finally paralysis of respiration and death occurs.
- D. When an injury occurs to the pons, motor or sensory paralysis of the third and fifth nerve may occur.
- E. When injury to a ventricle is present increased intracranial pressure resulting in choked disk ensues. Stertorous breathing, high temperature and convulsions accompanied by paralysis and rigidity of one side point to a hemorrhage into the lateral ventricle of the opposite side. (MacGruder.) Fractures of the base of the skull are apt to cause injury to one or more of the cranial nerves.

NERVES.

1. The olfactory nerve is injured by a fracture passing across the frontal fossa. Loss or derangement of the sense of smell results.

2. When the optic or second cranial nerve is involved, blindness occurs when it is directly severed. When the lesion occurs in front of the chiasm a *bitemporal* hemianopsia will result. A lesion back of the chiasm will cause a *bitemporal* hemianopsia. Lesions to the right or left of the chiasm cause incomplete homonymous hemianopsias (see White and Jelliffe) while lesions in the optic tract or occipital lobes will cause complete homonymous hemianopsia. When the lesion is in the occipital lobe, there may be, in addition to the above, an optic anosia. Exophthalmus may take place in fractures of the base from an arterio-venous aneurysm. (*Revista de la Asoc. Medica Argentina*, 1919, 30, 377.)

3. Motor Occuli. The motor oculi is especially apt to be involved in fracture of sphenoidal fossa. The third nerve supplies all the muscles of the eye, except the external rectus and the superior oblique. It also supplies the levator palpebra, the ciliary muscles and the contracting fibres of the pupil. (White and Jelliffe.) A fracture extending through the sphenoidal fossa is especially apt to cause disturbance in these structures.

4. Injury to the fourth nerve causes a diplopia and some dizziness when the patient looks downward and outward. In paralysis of the fourth nerve, it will be noticed that the upper eyelid of the affected side decends. However, it does so when the patient voluntarily closes his eyes.

5. The fifth nerve is most frequently involved in fractures at the base of the skull. This nerve contains both motor sensory fibres. The motor fibres supply the muscles of mastication. The sensory fibres supply the skin of the face, the mucous membrane of the superior, anterior posterior inferior nares, the teeth, the frontal, the ethmoidal and superior maxillary sinuses, the mucosa of the lips, the cheek, the posterior and portions of the mouth, the anterior two-thirds of the tongue and the tentorium cerebelli.

6. Sixth nerve. The eye on the affected side is rotated slightly outward in early stages of irritation following fracture. When the trauma to the nerve is severe or when paralysis, due to pressure occurs, there is inward rotation of the affected eye. The pupil is slightly smaller in the affected eye. (White and Jelliffe.)

7. Lesions of the seventh nerve. After its emergence from the pons, it lies in close relationship with the 5th and 8th nerves. When the lesion is cortical, occurring from a meningeal hemorrhage or depressed fracture in the mid-parietal region, the muscles of the lower part of the face are involved. The face is twisted to the sound side. The lips and angle of the mouth droop on the affected side. The nasolabial fold flattened. However, the eyes can be closed and the forehead wrinkled. The tongue, when protruded, is deflected to the paralyzed side.

If only a cortical irritation is present, convulsive movements of the face muscles occur. If the fracture is at the base of the skull and passes through the petrous part of the temporal bone, the seventh nerve may be involved in its passage through the bone. Lesions external to the bone cause, in addition to those enumerated for cortical lesions, paralysis of the muscles of the forehead. Also there is inability to close the eye on the affected side. If the lesion is in the stylo-mastoid foramen, there will be an impairment in taste in the anterior two-thirds of the tongue. Salivary secretion is also diminished. When to the

above, hyperacusis and tinnitus are added, the nerve further back in the canal is involved.

8. If the fracture is in the petrous portion of the temporal bone or in the posterior fossa of the skull, it may involve the eighth nerve. This is indicated by tinnitus and varying degrees of deafness. If the vestibular branch is involved vertigo may result.

The above nerves may be involved separately or in groups. When they are grouped they are combined as follows:

(a) When the 9th, 10th and 11th nerves are involved the fracture is probably a fracture through the foramina lacerum medium or pressure in the posterior fossa.

(b) When the 7th and 8th nerves are affected the fracture involves the internal auditory meatus.

(c) When the 2nd, 3rd, 4th and 6th nerves are affected the fracture involves the sphenomaxillary fissure.

(d) A lesion of the 5th nerve with dilatation of the pupils indicates the base of the sphenoid as the site of injury.

If the above groups are involved without other nerve derangements, it points rather definitely to the localization of a fracture through one of the foramina of exit. Sympathetic nerves or plexi may be involved. This gives rise to dilatation of the pupils of the affected side and flushing of the cheek of the affected side. Depending upon the extent and magnitude of the hemorrhage is the severity of the above symptoms.

X-ray.—A fracture of the skull can as a rule be diagnosed by a careful radiographic examination. A Bucky diaphragm gives a clear definition. Exposures should be made, especially to clearly define that portion of the skull which is opposite to the point of impact. A stereoscopic view is the best as it localizes the site and the direction of the fracture.

In the X-ray of a skull that is being examined for a suspected fracture neglect should not lead one to forget to examine the *suture lines* to see if they are sprung. Also examination of the *accessory sinuses* should be made to determine if there is any darkening due to a collection of blood.

The radiographic investigation has a negative as well as a positive value. This is well illustrated in a case I shall quote from Schiller. He found in a male 36 years of age, within two days after a blow with a club on the left temporal region, that there developed motor aphasia and Jacksonian epilepsy of the right extremity. A rupture of the middle meningeal artery was thought to be present. Röntgen examination revealed an angular fissure of the frontal bone, above the outer orbital edge and a separation of the coronal suture at the bregma,

the furrow of the middle meningeal artery was distinct and was interrupted. Operation for this reason was not performed. Complete healing occurred after two weeks. (See Plate 7 in Schiller's Monograph in the book by Lewandowsky.)

Remote symptoms resulting from fracture of the skull may be due to

I. *Pressure irritative cortical changes.*

(a) Motor cortical changes which may produce irritation and lead to interrupted explosive convulsive attacks of Jacksonian epilepsy.

The *modus operandi* of the production of motor reactions from a depressed fracture is as follows: During inspiration and expiration, there is a pulsation outward and inward of the brain, so that the dura is constantly impinging against the depressed bone, and there is constant irritation at the site of the fracture. In consequence of this osteophytic growth takes place (Urener). This in its turn causes sufficient cortical irritation to give rise to cortical convulsions. These convulsions we term epilepsy. Epilepsy may occur as a sequel to cerebral concussion, without any wound of the skull. It begins generally six to eight months after injury and may occur eighteen months to two years after injury, but it may appear shortly after the accident. (*Military Med. Annals*, p. 250.)

The location of injury may be in the frontal lobe, occipital lobe, rolandic area, cerebellar region and give rise to symptoms referable to these areas.

Continuous epilepsy is a condition described where there were spasmodic localized movements (4 or 5 a second) of extension and flexion in the left wrist, persisting during sleep, inability to separate or flex the fingers and inability to move the wrist and inability to use the thumb. In this case the fingers were semi-flexed and the thumb adducted. The form of objects were not recognized by the left hand. This condition followed a glancing bullet on the right parietal region. On trephining over the wound there was found an extradural hematoma. Patient had one or two attacks of Jacksonian epileptic seizures on the opposite side a day or two later. In three months patient returned to duty. This has been described as continuous epilepsy. (Polosson & E. J. Collets, *Brit. Med. Jour.*, 1915, p. 269.)

(b) Sensory cortical changes. Just as the cortical motor

centers may be affected by irritating or disturbing lesions, so too the sensory area may be involved and give rise to a syndrome described as Déjerine's Syndrome in which pain, heat and temperature are almost unaltered, but tactile sense of position and deep sensation, also bone and joint sensibility are lost with astereognosis. Hands are clumsy and ataxic. The lesion in the case described was found in the parietal region on the opposite side of the brain to the side involved and is located in the post central convolution, about the center of the Rolandic area, but more posterior, so that the stereognostic sense as it lies directly posterior to the sensory area was affected.

II. *Inflammatory reactive changes* may terminate as a meningitis or progress to abscess formation. Meningitis may be nonbacterial or bacterial. In the nonbacterial meningitis there is an œdema or serious infiltration of the pia-arachnoid. This œdema is so extensive at times that the arachnoid may be one-quarter to one-half inch thick. This pathological change is manifested by pain in the head (headache) localized as a rule to the area of the serous infiltration. There is localized tenderness to pressure and percussion.

When bacterial involvement of the meninges occurs the symptoms which follow are the result of the bacterial reactive inflammatory changes. These symptoms are:

1. Pain—Constant of gradually increasing severity generally localized to the area of involvement. Cephalic tenderness.
2. Stiffening of the neck muscles.
3. Elevation of temperature.
4. Changes in the composition and character of the cerebrospinal fluid which is under increased pressure and contains an increased number of cells of the polynuclear type. It also shows the presence of a considerable amount of globulin.
5. Signs of increased intracranial pressure, *i. e.*, slow pulse or a pulse rate slow in relation to the temperature. Vomiting may occur, restlessness, insomnia or sleep interrupted by loud cries may ensue. At times a choked disk may be found.

If the process continues to definite abscess formation the cerebral irritation symptoms cease and a remission may occur in the symptomatic reactions, so much so, that frequently the patient and attendants are lulled into false security. However, the cerebral tenderness per-

sists and dullness on percussion may be present. As the abscess increases the blood shows a greater polymorphonuclear increase (both proportional and total). Choked disk becomes more marked.

The pupil becomes dilated in the side of the abscess, then as the pressure increases both pupils may dilate and remain so. If one abscess is located near a motor area irritative convulsive movements may result in the related muscle group with a paralysis. If the destructive changes are of sufficient magnitude the final stage is one of gradually increasing pressure and exacerbation of all symptoms. The symptoms may not develop so in a regular progression as above. The transition from one group to another may be extremely rapid.

The above abscesses are of the subdural variety and presuppose an injury of the meninges with a solution of continuity of the bone and perhaps, but not necessarily, of the meninges. An abscess between the dura and the skull may arise from a blow on the head, without fracture or rupture of the continuity of the bone. The skin may become infected, the bone may later be involved and the infecting organisms may pass beneath the bone, between it and the dura. Werner reports such a case, in which the temperature continued high after drainage of a subcutaneous abscess, trephining of the bone finally evacuated an epidural abscess and the case went on to recovery.

New pathologic reactive changes in meninges or cortex may develop, such as

1. Scar tissues.
2. Chronic œdema of the pia-arachnoid is a condition which I have found in many cases which come to operation. The arachnoid appears as a thickened layer having somewhat the appearance of starch stiffening used in laundrying, as it is applied to the clothes. The arterioles could not be well defined. After this layer had been incised, it began to seep and in a short time considerable quantities of cerebral spinal fluid escaped. The membranes now flattened down on the cortex and the small blood-vessels became apparent. Normal pulsation started in the cortical vessels and pulsation of the brain became apparent. The absence of brain pulsation was, no doubt, due to an increase in the intracranial pressure. It is not likely that the small amount of œdema in the pia-arachnoid over

a local area would increase the general pressure to such an extent that the pulsation in the entire surface of the brain would cease, so that to account on the basis of pia-arachnoid œdema for increase of intracranial pressure, it would be necessary that the œdema be present over the greater part of the pia-arachnoid or else that the scar tissue present in the pia-arachnoid around the area of œdema made it impossible for the fluid to be drained. The constrictions on the venules by the same scar tissue accounting for the persistence of the œdema. This pressure is sufficient to cause headaches, localized to the area involved and in some instances epileptic attacks occur, the convulsive movements being confined to the muscles having their motor cortical centers in such an area. The skull over the area involved is tender to percussion and the patient may be apathetic and drowsy. In such cases great care should be exercised during operation not to rub the pia-arachnoid nor to irritate it in any manner. Several incisions should be made into it and even smaller pieces of the upper layer of the arachnoid cut away, in order that the edges may not unite and reproduce the old condition.

Cysts of the pia-arachnoid are a further development of the œdematous states or else are the result of the precipitation and organization of the solid elements of a blood clot and the persistence of the liquid contents as a cyst.

3. Cysts are especially marked in cerebrum after hemorrhage into the brain substance. Cysts may be
 - I. Epidural
 - (a) General or
 - (b) Circumscribed
 - II. Subdural
 - (a) Pia-arachnoid
 - (b) Cortical
 - (c) Infracortical, usually a cause of epileptiform convulsions.
4. Peripheral reactive changes around embedded foreign bodies.
5. Traumatic late apoplexy may occur according to White and Jelliffe. Signs of hemorrhage may develop even months after the trauma. (White and Jelliffe, p. 547; Refer to Stadel-

man—*Deutsch Med. Woch.*, 1903 and Allen-Jones, "Ment. and Nerve Dis.," 1909.)

6. Arterial aneurysm formation (Schiller, p. 149).
7. Calcification of the softened area of the brain.
8. Tumor of the brain (Schiller, p. 149). Schiller reports a case in which the cerebral symptoms could be traced to a severe trauma of the skull, years previously. As a residuum of the trauma there were deep depressions on the parietal bone at post-mortem a tumor of the brain was found.
9. New formations of bone at the site of fracture—at times a thick deposit of osteophytes at this point or a diffuse hyperostosis of the bone may occur (Schiller).

THE COEFFICIENT OF SAFETY IN GENERAL ANÆSTHESIA

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THE two words "safety" and "anæsthesia" are, in the light of present day knowledge, still quite incompatible. In order to increase the coefficient of safety and reduce the element of danger, a rather wide range of factors must be taken into consideration. These may be tabulated as follows:

1. Anæsthetic education.
2. Intrinsic characteristics of the anæsthetist.
3. The surroundings in which the anæsthesia is conducted.
4. The anæsthetic.
5. The method of administration.
6. The patient.

ANÆSTHETIC EDUCATION

There is a crying need of better instruction in anæsthesia in our American medical schools. The curriculum of the average school includes no such course, while in others the subject is merely wedged in, and forms a subsidiary of other branches of teaching. The laxity and indifference with which this, one of the most serious departments of medicine, is treated, is appalling and even inhumane.

We frankly must admit that the young graduate knows nothing about the administration of anæsthetics. Later on, during his internship, he gives a very limited number of anæsthesias, with practically no supervision, and with his attention directed mostly to the operation in progress. Thus lightly does he deal with sacred life entrusted to his care.

It so happens that not a few operations are entirely devoid of danger to life, but an anæsthesia always courts fatality and is omnipresent in each and every case. Patients have died of anæsthesia in the dental chair and during eversion of an ingrown toe-nail.

Status lymphaticus cases succumb before the operation is even commenced. Statistics of anæsthetic deaths in our larger hospitals are unnecessarily great and greater still because many more of them are ignorantly diagnosed as surgical shock, heart failure and embolism and are so classified. In some cases life is sacrificed because the choice of the anæsthetic itself has been unwise.

The so-called "nurse anæsthetist" is another evil influence which is mentioned only to be condemned. The presence of such a person in the operating room is an imposition and any arguments advanced in her behalf are simply begging the question. Nurses are unfit as anæsthetists as they lack the necessary preliminary education in anatomy, physiology, biology, chemistry, therapeutics and diagnosis. They subconsciously rest their responsibility upon the surgeon and he in turn assuming this burden, becomes in part, anæsthetist, and so oversteps his bounds.

Such facts as these make us stop and ponder, but reflection of this sort is excellent medicine. We can ill afford to face such criticism, just as this criticism may be.

As a means of correcting our serious shortcomings, and furthering scientific accuracy in this particular field, I would repeat a suggestion I have oft made. All medical schools should have a complete, well-systematized chair of Anæsthesia, presided over by a full-time professor. The course should be well-planned and be followed by well-balanced examinations. In the hospital interneship, the student should be under the strict guidance of the staff anæsthetist, and time should be set aside for this purpose. Later on, in general practice, if the physician encounters a questionable risk, he should enlist the services of a specialist. Every hospital should have on its staff a full-time anæsthetist, and where the size of the institution warrants, also an associate anæsthetist.

I believe that by following such a scheme as outlined, we could do much to relieve the present careless and negligent situation.

INTRINSIC CHARACTERISTICS OF THE ANÆSTHETIST

Not every one is adapted by virtue of personal characteristic to become a proficient and serious anæsthetist. Just what these characteristics are is difficult to define. It is an indescribable something within the psychology of the individual himself, a true personal

equation. He must not be of an excitable disposition. His nature should be calm and quiet, gentle yet firm and at all times should he be master of the situation. The fear of God should rest within him, and he should look upon every anæsthesia as an experiment with human life, the outcome of which cannot be foretold.

A thorough understanding of internal medicine, with special reference to diseases of the respiratory tract, cardio-vascular system, the liver, kidneys and nervous system, is an absolute essential.

The anæsthetist should never allow his attention to be distracted from his work regardless of what happens. He should never under any circumstances become absorbed in the operation nor allow himself to watch it, if only for an instant. His training should make him acquainted with the steps and stages of the operation, so that he may recognize them without looking and gauge the depth of his level of maintainance accordingly.

Responsibility is the unending key-note of the conscientious anæsthetist. This responsibility begins with the first introduction to the patient, perhaps several days before the operation, and does not terminate until several days after the operation when the subject has passed beyond all doubt of post-anæsthetic complications.

Another attribute required of the anæsthetist is mercy and tolerance. He must not be too proud, snobish or hasty to grant his patient the courtesy of a smile, a gentle touch of the hand or a few reassuring words. Trifling as these things may seem, they reflect the true spirit of Christ, and afford comfort and peace to a soul that is about to span the great bridge of oblivion and who knows whether or not it will ever return.

Alertness is another prerequisite. Alertness in detecting and correctly interpreting the slightest change in respiration, heart action, color, reflexes and tonus and this to be accomplished entirely subconsciously, without any visible effort. The anæsthetist must "feel" the condition of the patient.

Diligent study and research are elements which cannot be overestimated. They reflect the true character of the individual and show that his ideals are high and in keeping with the responsibility in which he is placed.

THE SURROUNDINGS IN WHICH THE ANÆSTHESIA IS CONDUCTED

Although this subject can be dismissed in a comparatively few words, its rôle, however, is far from being trivial. In a certain percentage of fatalities, the outcome would have been quite different under other surroundings.

The safest place to conduct anæsthesia is in the hospital operating room. The reason is obvious for here in time of need there are plenty of trained and willing hands. Means of resuscitation, such as the pulmotor, oxygen, the Farradic current, surgical assistance, intubation, the various stimulating drugs, and a table allowing the inverted position are at instant call. Other measures such as good light, plenty of hot and cold water, sufficient dry goods, cleanliness, quietness, and the exclusion of nervous and hysterical relatives or friends, play no small part as contributors to the element of safety.

Furthermore, in hospital practice, patients are as a rule anæsthetically prepared, whereas in the home or dental office especially, this preliminary precaution is overlooked. It may be said as a general axiom, that the further we stray from the hospital operating room, the greater becomes the "risque" of the administration.

THE ANÆSTHETIC

Our choice of a general anæsthetic is necessarily very limited as the list is small. The group includes ether, chloroform, mixtures of ether and chloroform with or without alcohol, nitrous oxide with or without oxygen, ethyl chloride, methyl chloride, and ethyl bromide. There are still others which have reached but an experimental stage, and been discarded as unsatisfactory for one reason or another.

Every general anæsthetic has its advantages and its draw-backs *per se* and these in turn become modified as they are applied to the patient, so that a given agent being hazardous to one class of cases, might well have the opposite effect on others, and in this delicate discrimination lies one of the greatest opportunities for increasing the coefficient of safety.

I shall not attempt to burden you with a consideration of the physical and chemical characteristics of the various anæsthetics nor to delve into the romantic histories surrounding them, as this does not lie within the scope of my discourse.

I am presenting the anæsthetics in order of greatest danger, considering those most lethal first.

ETHYL CHLORIDE

This is one of the most dangerous narcotics we possess. Collapse is more liable to follow ethyl chloride than any other anæsthetic. It is at times employed as a preliminary to ether, the change being made at the end of the stage of induction. Its use in complete anæsthesia, that is, through the stages of induction, maintenance and recovery, is to be discouraged.

One of the most frequent uses for this agent is as a substitute for nitrous oxide, because it is conveniently carried and comparatively cheap. Such a procedure is entirely unjustifiable in view of the much greater safety of nitrous oxide. In other words, it has been looked upon as a sort of glorified nitrous oxide which one could carry around in the vest pocket and use indiscriminately. The drug is highly toxic, produces a rapid anæsthesia, and is usually accompanied by masseteric spasm, thus necessitating placing a prop between the teeth before beginning its administration.

The dangers of ethyl chloride are twofold; first, death from simple overdose, and second, from asphyxia, as a result of spasm occurring in the respiratory tract. Anticipation of these difficulties will forestall untoward results. Thomas D. Luke reports twenty-three deaths in the short period of five years.

From the foregoing, it will be seen that ethyl chloride does not occupy a place in the armamentarium of the anæsthetist who is adverse to taking chances, and that he will substitute in its stead, nitrous oxide.

It seems opportune in this situation to say a few words about methyl chloride and ethyl bromide. Methyl chloride is very similar in every respect to ethyl chloride but is practically never used alone. It is found in the so-called "Somnoforme" which is a mixture of ethyl chloride 60 per cent., methyl chloride 35 per cent. and ethyl bromide 5 per cent. This mixture is now not much used.

Ethyl bromide is also similar to ethyl chloride, producing like the later drug, a rapid induction, stertorous breathing ensuing in about sixty to seventy seconds. It is used for very short operations, such as tooth extractions, adenoid curettage or tonsillotomies. It is unwise to repeat its administration at one sitting as may be done with ethyl chloride. Like ethyl chloride also it produces masseteric spasm and jactitation. Its use is attended with considerable danger, therefore being seldom employed.

CHLOROFORM

This is the next anæsthetic to present itself in the order of danger. Although chloroform is somewhat safer than ethyl chloride, it is still a very powerful and hazardous agent. It is probably the most frequently used anæsthetic, owing to its wide range of geographical distribution, being the drug of choice throughout the Western and Southern United States, Canada, Continental Europe, England, Scotland, Ireland and South America. It is only in the Eastern and Central United States that it has almost entirely given way to ether and even here in the rural districts, it is still extensively employed.

Chloroform was officially condemned by the Committee on Anæsthesia of the American Medical Association June, 15, 1912. In overdose, it kills in a concentration of 5 per cent. It is most dangerous during the stage of induction, at which time it is most commonly used, as for instance, preliminary to ether or in short incomplete anæsthesias.

One of the most noteworthy characteristics of chloroform is that it acts as a distinct protoplasmic poison, many of its evil effects not manifesting themselves until some time after the administration. These late symptoms are known as "delayed chloroform poisoning" and the condition is now quite generally recognized. It is the result of degenerative changes involving chiefly the liver and kidneys, the pathology in these organs closely resembling eclampsia, and consisting of congestion, hemorrhage, degeneration and necrosis.

The liver becomes yellow and fatty with hemorrhages often under the capsule and throughout its substance. There is also a central necrosis. The cells about the central vein disappear leaving a mass of granular material without nuclei or cell outline while nearer the periphery of the lobule, the cells are swollen and have undergone hyaline and fatty changes. A few normal liver cells may be found.

The kidneys show marked congestion, and are swollen, with occasional hemorrhages under the capsule, about the tubules, and in the pelvis. The cortex is thickened and markings indistinct. The cells of the tubules are swollen, granular, and loaded with fat. The lumen of the tubules is filled with granular material, fat globules, and coagulated serum.

The heart muscle may show fatty degeneration. From the foregoing, it is easily seen that chloroform is a distinct protoplasmic poison.

The admirable work of Bandler, Lengeman, and Leppman has proved that such changes do not occur with ether.

The symptoms of chloroform poisoning may develop from a few hours to a few days after the administration, and include progressive weakness, pallor or cyanosis, restlessness, vomiting, delirium, convulsions, stupor, coma, and death.

The causes of death during chloroform anæsthesia may be divided into those occurring during the three stages of anæsthesia. First, during induction, when a spasm of respiration occurs, and the drug continues to be poured on the mask, relief of the spasm being followed by several deep inspirations, and so a lethal dose suddenly entering the circulation. Also in this stage, a fatality may result from pneumogastric inhibition. Second, in the stage of maintenance, death may result from suddenly raising the head and shoulders causing a syncope which may develop into circulatory shock. Or in this stage, we may give simple overdose. Third, in the stage of recovery, a patient may die from progressive acidosis, the result of acute septicæmia or unrecognized diabetes.

One of the most striking characteristics of chloroform is to produce circulatory shock. I am personally not in favor of the so-called A.C.E. mixture or any chloroform mixtures for the reason that one is very likely to forget that they contain chloroform and become negligent in their use. They are, however, safer than chloroform alone, if given with caution. When the heart once stops in chloroform, it is usually a permanently damaged organ and very rarely yields to resuscitation.

Where chloroform is used, we may increase its coefficient of safety by following certain suggestions. These are—never to use it in acidosis or eclampsia, always to have the patient lying flat, to remove the mask during masseteric spasm, to use only fresh preparations, to admit plenty of air, never to lose the corneal reflex, and lastly never to change to chloroform after a prolonged unsuccessful ether induction.

Chloroform fatalities average about one in two thousand.

ETHER

From the standpoint of safety, ether is surpassed by only one other anæsthetic and that is nitrous oxide and oxygen. However, I feel that at the present time our observations of nitrous oxide and oxygen are not very extensive and the statistics far from accurate, so that eventually we may have occasion to alter our opinions, even in this direction.

Ether is the most disagreeable of all anæsthetics from the patient's point of view, the induction being slow, and recovery usually accompanied by retching, nausea, vomiting and headache. Its safety is its greatest virtue.

I wish to take this opportunity to criticize most emphatically the practice of heating ether vapor before administration. Owing to the high explosive nature of the drug, this procedure is most pernicious and several accidents and deaths are on record. The actual benefits derived from first warming the vapor are really insignificant and are ten-thousand-fold offset by the risk. I have long since discontinued this method in my practice.

Combining oxygen with ether (the so-called ether-oxygen anæsthesia) is the most recent method for increasing its standard of safety and also insures a more even and pleasant narcosis.

The chief element of safety in ether is that it acts as a distinct cardiac stimulant, being a derivative of alcohol. Heart action is increased in force and frequency and blood-pressure raised due to vasomotor stimulation. After prolonged administration however, it of course becomes depressant.

NITROUS OXIDE

Nitrous oxide alone can be used for only very short operations and is so employed mostly for dental extraction. Patients invariably become deeply cyanosed. If continued for more than two minutes, the patient's condition becomes critical from lack of oxygen. There is first cyanosis and jactitation followed later by asphyxia, as the small amount of oxygen admitted through atmospheric air is insufficient to maintain life. This method of administration is decidedly more unsafe than ether.

The method of combining oxygen with nitrous oxide is called nitrous oxide-oxygen or gas-oxygen anæsthesia and is the most recent and most signal contribution to the science of anæsthesia.

It is, of all anæsthetics, by far the most pleasant to take as it is practically devoid of odor and taste and does not give rise to suffocative attacks. The induction is very short, recovery rapid and seldom is there nausea and vomiting although these may occur in some cases. From point of view of the patient, it is the ideal anæsthetic. Its

greatest disadvantage, however, lies in the fact that it does not give complete relaxation in some instances and must therefore be supplemented by ether given through vaporization.

The elements of safety in the use of this drug are that it does not in any way irritate the respiratory tract, damage the kidneys or cause degenerative changes in the liver. Its effects on the cardio-vascular system are nil although at times and under certain pathological conditions, it does embarrass the circulatory system. Contrary to general belief, it does produce a slight increase in blood-pressure, but this is of itself insignificant.

Danger signals, which rarely occur during its administration, are prolonged and unremedial cyanosis, a slowing of the pulse rate and a peculiar whitish ring about the mouth. When these symptoms present themselves, excepting cyanosis, they appear very suddenly somewhat as in chloroform.

The anæsthetic balance in nitrous oxide-oxygen anæsthesia is extremely delicate, the very slightest change in administration causing immediate reaction. It is the most difficult anæsthetic to administer.

I wish to urge most emphatically that the patient himself must at all times be the criterion upon which the proportion of gases delivered is based. This is of the utmost importance. Where this rule is disregarded, it invariably results in an unsuccessful anæsthesia, at times greatly endangering the life of the patient and always extremely embarrassing the anæsthetist, and causing no end of annoyance to the surgeon. I make special mention of this fact, because a great many of the gas-oxygen apparatuses on the market are adjustable on a scale of theoretical and so-called scientific percentages. Nothing could be more fallacious, absurd and truly unscientific, as each patient presents different and even constantly varying requirements. Recognizing the demands of the patient and employing one's skill in meeting them by properly proportioning the mixture of gases is the true test of efficiency and is the very element which makes the administration of nitrous oxide and oxygen so difficult.

Nitrous oxide and oxygen is sometimes combined with chloroform or ethyl chloride. This practice detracts from its safety. A mixture of gas-oxygen and ether on the other hand is without such risk and in the average case proves very satisfactory where greater relaxation is desired.

THE METHOD OF ADMINISTRATION

The choice of a suitable method for administering an anæsthetic is a potent element in contributing to either the safety or danger of the patient. The virtues of a well-chosen anæsthetic are frequently counterbalanced by an improper form of administration. The following remarks will help materially to raise the coefficient of safety.

Ethyl chloride may be given either by the open or closed methods. The closed method is more satisfactory and is carried on by means of the rebreathing bag. When stertor results free air should be administered. It is unsafe to use more than 4 c.c. of the drug. Loss of the lid reflex represents the limit of safety and abolished corneal reflex with dilated pupil should not be allowed.

Ethyl bromide is best given by the open drop method. Exclusion of air is not in the interest of safety. The legitimate dose varies from one to three drams.

Chloroform may be given in only one way and that is by the open drop method with ample admixture of free air. It should never under any circumstances whatever be given by any form of closed administration, such as the closed cone or rebreathing bag. The secret of a tranquil chloroform anæsthesia depends upon admitting a high percentage of free air and this is accomplished by holding the open mask at least one inch distant from the face.

Ether, which is next in order, permits of greater variation in mode of administration than any other anæsthetic. It may be given in the following ways: Open drop, semi-open drop, closed drop, the old-fashioned closed cone, oral insufflation, intrapharyngeal insufflation, intratracheal insufflation, per rectum and intravenous. A study of these various methods is complex and exhaustive, therefore I shall not take time to consider them here but will merely elucidate the advantages of those most important.

The advantages of the open drop method are that the apparatus is simple, and plenty of oxygen is always admitted; it is best for straight ether inductions, and is very applicable to children. Its disadvantages are that it tends to acapnia, increases loss of body heat, is very wasteful of ether, and is not so satisfactory where morphine has first been given. The semi-open drop has essentially the same foregoing characteristics, except that they are somewhat modified.

The closed drop, which is accomplished by means of a rebreathing

bag, is less wasteful of ether, tends to hypercapnia, preserves body heat, allows of a more delicate balance, gives a speedier induction and tends to overcome the slow and shallow respiration incident to preliminary morphine injection.

The closed cone is so unscientific, cruel and obsolete that it deserves no mention. Its use is admission of extreme anæsthetic ignorance.

Oral insufflation consists of delivering ether vapor with or without oxygen to the oral cavity. Its advantage lies in delicacy of control.

The intrapharyngeal and intratracheal methods are very similar excepting for anatomical difference. Ether is delivered in vaporized form by means of a very complex apparatus called an anæsthetometer. The vapor enters through catheters. These methods are of advantage in operations about the head and neck. They are exceedingly technical and can be conducted only by one very expert. Their use should be limited to cases where they are especially demanded. They all tend to lessen the coefficient of safety.

Ether per rectum may be given in oliveoil or combined with oxygen. The latter method, that is, with oxygen, is nearly obsolete. Without entering into discussion, I merely wish to state that the procedure is dangerous and should be employed only by the expert anæsthetist. It is suitable for the same class of cases as intratracheal insufflation and does not require the complex and expensive apparatus, the only requirement being a rectal tube and a funnel. Its disadvantages are its danger, that it is unreliable, that it may cause distention of the rectum with proctitis, that injections causing untoward symptoms cannot well be recovered and that in obese individuals it may interfere with respiration.

The administration of ether by intravenous injection consists of introducing a solution of about 5 per cent. ether in normal salt or Ringler's solution, into one of the veins at the bend of the elbow by means of an irrigator and canula. The method requires some surgical technic and strict asepsis. The anæsthetic control of the patient is most delicate, recovery occurring almost immediately upon cessation of administration.

Needless to say the method requires an expert.

Like all other complicated anæsthesias, this form has certain disadvantages. These are as follows: Preparation of the patient consumes considerable time; there is an increase of blood-pressure;

it increases hemorrhage; there is a possibility of septic thrombosis as in any venous operation, and lastly, it has in some instances produced pulmonary eclema.

Nitrous oxide or nitrous oxide and oxygen can be administered in only one way and that is by the absolute closed method. The method is dependent upon rebreathing and it is prerequisite that the face piece admit no leakage of air, otherwise the patient cannot be carried beyond the stage of induction. All such apparatuses are equipped with a ventilating chimney by which the depth of rebreathing may be controlled. If the stage of maintenance is to be prolonged, it is essential to precede the administration by morphine and atropine or morphine and hyoscine. My personal preference inclines toward hyoscine.

This form of anæsthesia greatly increases our coefficient of safety.

THE PATIENT

Patients presenting themselves for anæsthesia exhibit various manifestations of disease, idiosyncrosy and environmental influence, all of which must be carefully considered. Some subjects contraindicate anæsthesia altogether, as for example status lymphaticus, advanced diabetes or very well-marked acidosis from any cause. If the acidosis be slight, the hazard of the anæsthetic can be greatly reduced by proper preparatory treatment beginning several days before, including the administration of sodium bicarbonate in large doses, and choosing nitrous oxide as the narcotic.

Cases with irritation in the respiratory tract such as bronchitis, pneumonia and tuberculosis are better treated by chloroform than ether, if nitrous oxide cannot be had. Ether undoubtedly causes an exacerbation of a quiescent tubercular focus in the lung and is also very apt to convert an innocent bronchitis to a serious pneumonia. In this class of cases chloroform does find its true level.

Myocarditis and any form of cardiac weakness are indications for the use of ether inasmuch as this drug, being an alcohol derivative, is a cardio-vascular stimulant. I prefer it to nitrous oxide in such cases. Chloroform here is condemned.

Nephritis, in varying form and degree, should receive nitrous oxide as first choice.

Unfortunately, our patients very often present a variety of afflic-

tions so that an anæsthetic well chosen for one condition would be contraindicated by another and here one must choose the lesser of two evils.

Where the risk of a general anæsthetic is considerable, the situation may very often be well handled by combining local analgesia with amnesia. However, this is not within the scope of my present subject.

Nervous patients, those with a neurotic tendency, often give the anæsthetist a deal of trouble and even anxiety. Although they are not dangerous as a class, their reactions (especially pupillary) and reflexes are so altered, exaggerated and uncertain and their general behavior throughout the entire anæsthesia so peculiar, that it is often extremely difficult to recognize their exact anæsthetic status. It is very desirable to visit these patients a few times before their operation and employ every known means to produce mental quietude, even to the administration of sedatives.

Obese patients with short necks are often difficult to handle, although the condition is hardly ever serious.

Asthmatic cases may or may not become dangerous, but always require unusual care.

Persons with a goitre should receive as little fright and shock as possible.

In closing I should like to emphasize that anæsthesia is a distinct science, not limited to the mere administration but extending farther into the general field of medicine than any other specialty. Allow me furthermore to introduce a plea for improved education.

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